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Supreme Court, U.S.
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No.

IN THE

Supreme Court of the United States

JEFF TAMRAZ AND TERRY TAMRAZ, PETITIONERS

v.

LINCOLN ELECTRIC COMPANY; HOBART BROTHERS COMPANY; ESAB GROUP, INC. AND TDY INDUSTRIES, INC.

PETITION FOR A WRIT OF CERTIORARI TO THE UNITED STATES COURT OF APPEALS FOR THE SIXTH CIRCUIT

PETITION FOR WRIT OF CERTIORARI

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QUESTIONS PRESENTED

I. Under *Daubert's* flexible standard of admissibility, must an expert testifying about the cause of petitioner's parkinsonism prove every inference and assumption supporting his opinion?

II. Where a majority of the court of appeals employs a *de novo* standard of review instead of an abuse of discretion standard to reverse the district judge's admission of expert opinion about the cause of petitioner's parkinsonism, does it usurp the district court's "gatekeeping" function under *Daubert* and undermine *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999) and *General Electric Co. v. Joiner*, 522 U.S. 136 (1997)?

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OPINIONS BELOW

The published opinion of the United States Court of Appeals for the Sixth Circuit in *Jeff Tamraz and Terry Tamraz v. Lincoln Electric Company, et al.*, C.A. Nos. 08-4015/4016, decided September 8, 2010, reported at 620 F.3d 665 (6th Cir. 2010), reversing the District Court's denial of respondents' motions for judgment in their favor as a matter of law under Fed. R. Civ. P. 50(b) and remanding for a new trial, is set forth in the Appendix hereto (App. 1-42).

The unpublished decision of the District Court for the Northern District of Ohio, Eastern Division, in *Jeff Tamraz and Terry Tamraz v. Lincoln Electric Company, et al.*, Docket No. 1:04-CV-18948, decided July 18, 2008, denying respondents' post-trial motion for judgment in their favor as a matter of law under Fed. R. Civ. P. 50(b), is set forth in the Appendix hereto (App. 43-126).

The unpublished transcribed decision of the District Court for the Northern District of Ohio, Eastern Division, in *Jeff Tamraz and Terry Tamraz v. Lincoln Electric Company, et al.*, Docket No. 1:04-CV-18948, dated November 1, 2007, denying respondents' pre-trial motion to exclude from trial the testimony of petitioners' expert, is set forth in the Appendix hereto (App. 128-132).

The unpublished Order of the United States Court of Appeals for the Sixth Circuit in *Jeff Tamraz and Terry Tamraz v. Lincoln Electric Company et al.*, C.A. Nos. 08-4015/4016, filed November 9, 2010, denying petitioners' petition for rehearing or, in the

alternative, for rehearing *en banc*, is set forth in the Appendix hereto (App. 127).

JURISDICTION

The majority decision of the United States Court of Appeals for the Sixth Circuit reversing the District Court's denial of respondents' motions for judgment in their favor as a matter of law under Fed. R. Civ. P. 50(b) and remanding the case for a new trial, was decided on September 8, 2010; and its further order denying petitioners' timely-filed petition for rehearing or, in the alternative, for rehearing *en banc* was issued on November 9, 2010 (App. 1-42;127).

On January 28, 2011, this Court, Kagan, J., granted petitioners' application for an extension of time to file their petition until March 9, 2011 (Docket No. 10A744).

This petition for writ of certiorari is filed within the time allowed by Justice Kagan's order permitting petitioners to file their petition by March 9, 2011.

The jurisdiction of this Court is invoked pursuant to the provisions of 28 U.S.C. § 1254(1).

RELEVANT PROVISIONS INVOLVED

28 U.S.C. § 1332(a)(1):

> **(a)** The district courts shall have original jurisdiction of any civil actions where the matter in controversy exceeds the sum of $75,000.00, exclusive of interests and costs, and is between--

(1) citizens of different States....

Fed. R. Civ. P. 50(a), (b) and (e):

(a) Judgment as a Matter of Law.

(1) ***In General.*** If a party has been fully heard on an issue during a jury trial and the court finds that a reasonable jury would not have a legally sufficient evidentiary basis to find for the party on that issue, the court may:

(A) resolve the issue against the party; and

(B) grant a motion for judgment as a matter of law against the party on a claim or defense that, under the controlling law, can be maintained or defeated only with a favorable finding on that issue.

(2) ***Motion.*** A motion for judgment as a matter of law may be made at any time before the case is submitted to the jury. The motion must specify the judgment sought and the law and the facts that entitle a movant to the judgment.

(b) Renewing the Motion After Trial; Alternative Motion for a New Trial. If the court does not grant a motion for judgment as a matter of law made under Rule 50(a), the court is considered to have submitted the action to the jury subject to the court's later deciding the legal questions raised by the motion. No later than 28 days after the entry of judgment—or if the motion addresses a jury issue not decided by a verdict, no later than 28 days after the jury was discharged----the movant may file a renewed motion for judgment as a matter of law and may include an alternative or joint request

for a new trial under Rule 59. In ruling on the renewed motion, the court may:

(1) allow judgment on the verdict, if the jury returned a a verdict;

(2) order a new trial; or

(3) direct the entry of judgment as a matter of law.

....

(e) Denying the Motion for Judgment as a Matter of Law; Reversal on Appeal. If the court denies the motion for judgment as a matter of law, the prevailing party may, as appellee, assert grounds entitling it to a new trial should the appellate court conclude that the trial court erred in denying the motion. If the appellate court reverses the judgment, it may order a new trial, direct the trial court to determine whether a new trial should be granted, or direct the entry of judgment.

Fed. R. Evid. 104(a) and (b):

(a) Questions of admissibility generally. Preliminary questions concerning the qualification of a person to be a witness, the existence of a privilege, or the admissibility of evidence shall be determined by the court, subject to the provisions of subdivision (b). In making its determination it is not bound by the rules of evidence except those with respect to privileges.

(b) Relevancy conditioned on fact. When the relevancy of evidence depends upon the fulfillment of a condition of fact, the court shall admit it upon, or subject to, the

introduction of evidence sufficient to support a finding of the fulfillment of the condition.

Fed. R. Evid. 702:

Testimony By Experts.
If scientific, technical, or other specialized knowledge will assist the trier of fact, to understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Fed. R. Evid. 703:

Bases of Opinion Testimony By Experts.
The facts or data in the particular case upon which an expert bases an opinion or inference may be those perceived by or made known to the expert at or before the hearing. If of a type reasonably relied upon by experts in the particular field in forming opinions or inferences upon the subject, the facts or data need not be admissible in evidence in order for the opinion or inference to be admitted. Facts or data that are otherwise inadmissible shall not be disclosed to the jury by the proponent of the opinion or inference unless the court determines that their probative value in assisting the jury to evaluate

the expert's opinion substantially outweighs their prejudicial effect.

STATEMENT

Petitioner Jeff Tamraz ("petitioner" or "Tamraz") worked as a welder from 1979 to 2004. He spent most of his 25-year career working in California on many structures that are part of the San Francisco skyline. An accomplished welder with 18 welding certifications, Tamraz was in high demand. The welding consumables provided to Tamraz throughout his welding career produce fumes containing manganese which when inhaled could cause permanent neurological injury or brain damage which can leave a welder permanently and seriously disabled. Yet respondents never apprised Tamraz of this fact; his appreciation of the hazards of welding fumes was limited to welding around asbestos or welding on metals coated with lead-based paint, at which times he occasionally was instructed to use a respirator.

Tamraz used welding consumables called "flux-cored wire," one of the smokiest welding materials which produce fumes with one of the highest concentrations of manganese. He welded in confined spaces, underground and in shipyards. At times, the smoke from welding would "literally cover his safety lens charcoal black." (J.A.706-707).

Around 2000-2001, Tamraz, then just 44 years of age, began experiencing neurological symptoms that became so severe that he could not care for himself. Once a strong, tireless ironworker, Tamraz had tremors, loss of coordination, drooling and slowed

movements. He suffered fatigue, depression, poor sleep and lack of sex drive; he exhibited a mask-like face; he spoke in whispers and his muscles were stiff and rigid. In May 2004, neurologist Dr. Michael Siegel diagnosed Tamraz as suffering from parkinsonism, i.e., a neurological syndrome characterized by tremor, bradykinesia (slow movement), rigidity and gait disturbance.

The underlying causes of Parkinsonisms can be categorized as: (1) secondary parkinsonism brought about by an insult to the brain such as exposure to toxic drugs or chemicals, including manganese; (2) parkinsonism-plus syndromes where the patient exhibits some neurological manifestation in addition to the classic symptoms of parkinsonism; and (3) idiopathic Parkinson's Disease, the bulk of neurodegenerative conditions with no known cause or etiology. Dr. Siegel initially diagnosed Tamraz as suffering from parkinsonism, probably caused by his long-term exposure as a welder to manganese.

In September 2004, Tamraz and his wife Terry Tamraz ("petitioners") brought suit in the United States District Court for the Northern District of Ohio against Lincoln Electric Company, Hobart Brothers Company, ESAB Group, Inc. and TDY Industries, Inc. ("respondents"), as well as BOC Group, Inc., all of whom provided the welding materials Tamraz used during his welding career. Positing federal jurisdiction based on diversity of citizenship, petitioners alleged that the fumes from respondents' products contained manganese that caused his neurological condition and that the labels on these products failed to warn him of the danger.

Tamraz's case was consolidated with ongoing multidistrict litigation in the Northern District of Ohio, *In re: Welding Fume Prods. Liab. Litig.*, No. 03-cv-17000, MDL No. 1535. District Judge Kathleen M. O'Malley, fulfilling her "gatekeeping" function under *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), held three weeks of hearings in 2005 in order to assess the reliability and methodologies of proffered expert opinion testimony linking welding fume exposure to parkinsonism syndromes including Parkinson's Disease.

The district judge heard extensive testimony from "world-class experts" of the neurological community about the methods they use to make diagnoses of the various forms of parkinsonism, including manganese-induced parkinsonism, and whether there was a sufficient basis to support a claim that manganese could cause Parkinson's Disease. Experts on both sides agreed that the principal method they employ in order to discover what disease process is causing a patient's symptoms is "differential diagnosis," a process by which the physician considers all relevant potential causes of the symptoms and then eliminates alternative causes based upon a physical examination, clinical tests and a thorough case history.

At the completion of these *Daubert* hearings, Judge O'Malley issued a comprehensive 82-page order denying respondents' motion to exclude expert opinion testimony that welding fume exposure causes Parkinson's Disease. (J.A.85-166). She concluded *inter alia* that while clinical symptomatology is the tool most often used by practicing neurologists to make differential diagnoses between Parkinson's Disease

("PD") and manganese-induced parkinsonism ("MIP"), the symptoms for both diseases cannot be pigeon-holed neatly into one category or the other, as respondents urged. (J.A.144-148). These symptoms overlap so that "a person suffering from MIP will sometimes fit within the clinical definition of PD." (J.A.146-147).

Finding that these plaintiffs had adduced at least two epidemiological and eleven non-epidemiological lines of evidence----some including respondents' own experts—showing that welding and its attendant exposure to manganese can cause the onset of a parkinsonian syndrome that is similar to and can be diagnosed as PD, the district judge concluded that "the sum of epidemiological and other evidence proffered by the parties [is] sufficiently reliable to support the assertion that exposure to welding fumes can cause, contribute to, or accelerate a parkinsonian syndrome that some doctors will diagnose as PD." (J.A.162-166).

On March 21, 2007, and again in June 2007, Tamraz's treating physician, Dr. Walter Carlini, an experienced, board-certified neurologist, examined Tamraz and took his medical/occupational history, both accepted techniques in the neurological community for forming a differential diagnosis to discover what disease process was causing Tamraz's symptoms. Based on this information, together with his general knowledge and experience about movement disorders, Dr. Carlini diagnosed Tamraz-----a veteran welder of 25 years working in closely confined spaces with heavy exposure to manganese fumes who experienced the onset of profound neurological injuries at the age of just 44----as suffering from manganese-induced parkinsonism.

As Dr. Carlini reasoned during his videotaped testimony, Tamraz exhibited symptoms closer to Parkinson's Disease than those of manganism, the most egregious manifestation of brain damage resulting from severe manganese overexposure; his symptoms were more consistent with lower-dose, long-term manganese exposure. Moreover, because Tamraz experienced "early onset" parkinsonism at the age of just 44, Dr. Carlini believed that Tamraz "*most likely* has a genetic predisposition to Parkinson's Disease and that [his long term exposure to] the manganese tipped him over" into parkinsonism. (J.A.601;604)(emphasis supplied). He held this diagnosis of manganese-induced or manganese-triggered parkinsonism with a reasonable degree of medical certainty "because that seemed the most likely explanation for [Tamraz's] early onset parkinsonism," a key factor in his diagnosis. (J.A.601;604;758).

The district court selected Tamraz's case for one of several bellwether trials to guide the resolution of the other trials. (App. 3). Before trial, respondents moved to exclude parts of Dr. Carlini's testimony. (App. 128-132). Judge O'Malley denied the motion:

> ....I have read all the briefs. I have read...the two depositions that were taken of Dr. Carlini. I have gone back and reread the Court's *Daubert* opinion...and I have decided that I am going to deny [respondents'] motion.
>
> To a large extent, [respondents'] motion asks the Court to draw bright lines regarding diagnoses of movement disorders that I have already declined to draw, and I have already

> decided that the current state of the science does not require to be drawn.
>
> ....I see nothing lacking in [Dr. Carlini's] qualifications to render opinions regarding movement disorders, and *I see nothing in his methodology which is either flawed or inconsistent with the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used and have described as appropriate diagnostic methods.*
>
> I think it is telling that the defendants have not proffered an affidavit from any expert indicating that there is anything wrong with Dr. Carlini's methodology, or that there is anything faulty with respect to his qualifications....It is clear that the defendants have fair grounds to attack the somewhat unusual diagnosis that Dr. Carlini renders in this case...but that to me *goes to the weight and not the admissibility of his testimony.*

(App. 130-132)(emphasis supplied).

An eleven-day jury trial ensued in the district court. Tamraz's primary expert witness, Dr. Paul Nausieda, testified that Tamraz suffered from manganese-induced parkinsonism. (App. 29). Dr. Siegel, Tamraz's former treating neurologist who had initially diagnosed him with a form of Parkinson's Disease due to manganese poisoning, now testified that while he could not rule out this possibility, Tamraz likely suffered from parkinsonism resulting from factors other than manganese exposure. (App. 29-30). The jury heard Dr. Carlini's videotaped testimony that Tamraz likely

had a genetic predisposition to Parkinson's Disease and his long-term exposure to manganese as a veteran welder of 25 years working in closely confined spaces without a respirator triggered his underlying predisposition and tipped him into Parkinson's Disease. (App. 30). Finally, respondents' lead expert, Dr. Anthony Lang, admitted that exposure to manganese can cause parkinsonism but he did not believe that Tamraz's parkinsonism was caused by manganese exposure. (App. 29).

After the jury found respondents liable on the basis of strict liability and negligent failure to warn, awarding petitioners $20.5 million in damages, respondents filed motions under Fed. R. Civ. P. 50(b) to overturn the verdict claiming, *inter alia*, that Dr. Carlini's testimony was speculative and not based on published literature or scientific studies. (App. 3;30;43-126). Judge O'Malley granted the defendant BOC Group, Inc.'s motion because Tamraz had failed to show that he had used welding consumables manufactured by this defendant. (App. 44; 88-90). She denied respondents' post-trial motions. (App. 44-45; 53-63).

The district judge adverted to evidence adduced at trial showing that the welding industry, respondents among them, were well aware of the dangers of manganese in welding fumes at least as early as the start of Tamraz's welding career; that Tamraz regularly was exposed without a respirator and in confined spaces to welding products that emitted fumes containing high manganese levels; that respondents themselves conceded that exposure to manganese in welding fumes can in some circumstances cause manganese-induced parkinsonism; that the symptoms

of manganese-induced parkinsonism are similar to those of idiopathic Parkinson's Disease; and that individual susceptibility to the adverse effects of manganese varies considerably. (App. 54-63). She therefore "easily conclude[d] that there was presented at trial substantial evidence to support the opinions of Mr. Tamraz's experts that his exposure to the fumes given off by [respondents'] products was sufficient to, and did, cause his injuries, with reasonable medical probability" and noted that respondents had vigorously cross-examined Dr. Carlini and provided contrary evidence on disputed points. (App. 62).

Respondents appealed and a majority of the court of appeals determined that Dr. Carlini's opinion testimony that respondents' welding products triggered manganese-induced parkinsonism in Tamraz does not satisfy the requirements of Fed. R. Evid. 702. (App. 1-27). While it agreed with Dr. Carlini that Tamraz does suffer from some form of parkinsonism, the majority characterized his further opinion that Tamraz's parkinsonism was "manganese-induced" as nothing more than a working hypothesis on his part and not admissible scientific "knowledge" under Rule 702.(App. 6; 8). As the majority saw it, "Rule 702 requires more than subjective belief or unsupported speculation and Dr. Carlini's opinion therefore should have been excluded." (App. 8-9, quoting *Daubert*, 509 U.S. at 590).

According to the majority, in order to reach his opinion, Dr. Carlini engaged in a seven-step analysis that: (1) Tamraz was exposed to welding fumes "presumably" containing manganese; (2) he developed symptoms of Parkinson's Disease not approaching the

severity of symptoms of manganism; (3) the role of genetic factors that cause some form of otherwise idiopathic Parkinson's Disease is still under study; (4) some literature has hypothesized that toxins combined with genetics may cause other cases of Parkinson's Disease; (5) manganese is known to cause manganism, so it would be a possible candidate for triggering Parkinson's Disease as well;(6) Tamraz may have the genes for Parkinson's Disease; and (7) manganese may have triggered these genes and given Tamraz parkinsonism. (App. 9).

The majority thought that the last four steps of Dr. Carlini's analysis were nothing more than "speculative jumps" bereft of "knowledge" or "sufficient facts or data" or the "product of reliable principles and methods...applied reliably to the facts of the case," as required by Rule 702. (App. 9-10). It found the scientific literature relied upon by Dr. Carlini to form a link between environmental toxins such as manganese and a genetic predisposition to Parkinson's Disease was all "theoretical;" that he knew of no studies finding a link between manganese and Parkinson's Disease; and that he was guessing about Tamraz's underlying predisposition to Parkinson's Disease because Tamraz had no family history of Parkinson's Disease. (App. 10). The majority thus concluded that Dr. Carlini's opinion was "speculative three times over."(*Id.*).

Furthermore, it believed that Dr. Carlini's final step of concluding that manganese likely caused Tamraz's condition failed to account for the fact that Parkinson's Disease occurs in the general population usually without an identified cause, making it hard to

attribute Tamraz's case to manganese over all of the other possible causes, especially when the probability of getting parkinsonism from an underlying predisposition is unknown. (App. 10-11). Nor did the majority think that Dr. Carlini's reliance on Tamraz's very early onset of the disease was justification for concluding that manganese caused his parkinsonism since ten percent of persons with Parkinson's Disease develop symptoms before age 50, a fact that "does not create an inference that something unusual must have caused it in Tamraz."(App. 11-12).

In reaching this result, the majority thought that Dr. Carlini as a treating neurologist focusing on diagnosis may well think about etiology differently from the way judges and juries do and that this "low threshold for making a decision serves well in the clinic but not in the courtroom, where decision requires not just an educated hunch but at least a preponderance of the evidence." (App. 16-17). His "differential diagnosis" of Tamraz fails, according to the majority, because his efforts to "rule in" manganese exposure as a possible cause or to "rule out" other possible causes rest on speculation, not a valid methodology. (App. 18-19). Because Dr. Carlini was the only expert who testified that Tamraz has Parkinson's Disease caused by manganese, his testimony was not harmless and the majority reversed the district court's decision admitting this testimony and remanded the case for a new trial. (App. 22-26).

Circuit Judge Boyce Martin dissented. (App. 27-42). He first criticized the majority for paying lip service to the abuse of discretion standard of review mandated by *Kumho Tire Co., Ltd. v. Carmichael*, 526

U.S. 137 (1999), while, in fact, applying a *de novo* standard that substitutes the majority's own opinion for that of the district judge, who conscientiously conducted three weeks of *Daubert* hearings in this multi-district litigation in order to assess the opinions and methodologies of competing expert witnesses about the causal connection between manganese and parkinsonism. (App. 27-33). The proper inquiry----*never* pursued by the majority----was whether Judge O'Malley abused her broad discretion as the trial judge in determining that Dr. Carlini's expert testimony was not inadmissible "junk science" but rather testimony falling within the "range where experts might reasonably differ." (App. 30-31).

As the dissent saw it, under this deferential standard, an appellate court should interfere only where it is "absolutely clear" that the testimony is nothing more than "junk science" which a jury cannot be trusted to evaluate and that its admission is therefore arbitrary, unjustifiable or clearly unreasonable. (App. 31-33). After all, the underlying concern which *Daubert* and its progeny sought to address was the exclusion of "junk science," *not* the exclusion of expert testimony within a "range where experts might reasonably differ." (App. 33; 35). Because Judge O'Malley, as *Daubert's* "gatekeeper," reasonably evaluated Dr. Carlini's testimony in light of the broad range of expert opinions and found that it was sufficiently reliable to be admissible----in fact, she found that his expert testimony relies on "the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used"----the dissent concluded that the district judge did not abuse her discretion by admitting it. (App. 32-33).

Moreover, Judge Martin took issue with the majority's assertion of gaps and speculation in Dr. Carlini's analysis.(App. 36-41). At the time of his testimony in 2007, there was a lively debate, "theoretical" or not, in the published literature and among his peers in the neurological community about the connection between exposure to manganese and parkinsonism; and there was scientific testing of individuals exposed to manganese who later developed Parkinson's Disease, satisfying one, if not several, of *Daubert's* requirements.(App. 37-38).

Even more fundamental, the dissent believed the majority's insistence on scientific testimony without gaps of unprovable inferences or assumptions misconceives the search for scientific truth:

> ...[W]e must be cautious in demanding the type of finality from science that we have come to expect in law..., especially when considering new cases of newer scientific studies....[where] the science itself may be incapable of absolute proof.
>
> ....The fact that scientists have not reached consensus regarding medical causation does not render reliance on a scientist's theory improper expert testimony, particularly when, as in this case, the expert is relying on studies that appear to have been conducted *using standard methodology.*
>
> ....Because Dr. Carlini *relied on scientific methodology used by other experts in his field...*, I do not believe the district court abused its discretion in admitting his testimony....What weight to grant his testimony was a question for

> the jury, not an appellate court sitting far removed from the trial.

(App. 40-41)(emphasis supplied).

On November 9, 2010, the court of appeals denied petitioners' timely-filed petition for rehearing or, in the alternative, for rehearing *en banc*. (App. 127). On January 28, 2011, this Court, Kagan, J., granted petitioners' application for an extension of time to file their petition until March 9, 2011. (Docket No. 10A744).

REASONS FOR GRANTING THE PETITION

I. By Demanding That An Expert Who Testifies About The Cause of Petitioner's Parkinsonism Prove Every Inference And Assumption Contained In His Opinion, The Majority Nullifies *Daubert's* Flexible Standard For Admitting Such Testimony And Conflicts Specifically With The Decisions Of At Least Two Sister Circuits.

Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579 (1993) empowered district courts to act as "gatekeepers" to protect juries from misleading or unreliable expert testimony by assessing the reliability of the principles and methodologies used to reach the expert's opinion. *Id.* at 589-592. It adopted a two-step inquiry for the admissibility of scientific evidence contained in the Federal Rules of Evidence 104(a) and 702. *Id.* at 592-593.

First, the district court determines if the expert's proffered testimony----whether it concerns

scientific, technical or other special knowledge----has "a reliable basis in the knowledge and experience of his [or her] discipline." *Id.* at 592. See also *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 149 (1999). By conducting a preliminary inquiry into the expert's qualifications and the admissibility of proffered evidence, a district court fulfills its initial obligation under Rule 104(a) ("Preliminary questions concerning the qualification of a person to be a witness...or the admissibility of evidence shall be determined by the court."). In this initial inquiry, the district court determines "whether the reasoning or methodology underlying the testimony is scientifically valid and...whether that reasoning or methodology properly can be applied to the facts in issue." *Id.* at 592-593. Expert testimony must be reliable under Rule 702 and the district judge can consider, *inter alia*, whether a theory has been or can be tested or falsified; whether the theory or technique has been subject to peer review and publication; whether there are known or potential rates of error with regard to specific techniques; and whether the theory or approach has "general acceptance." *Id.* at 593-594.

The purpose of this inquiry is to ensure that an expert "employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Kumho Tire Co.*, 526 U.S. 150;152-153. As the *Daubert* Court concluded,

> [t]he inquiry envisioned by Rule 702 is...a flexible one. Its overarching subject is the scientific validity---and thus the evidentiary relevance and reliability---of the principles that underlie a proposed submission. The focus, of

> course, must be solely on principles and methodology, not on the conclusions that they generate.

509 U.S. at 594-595(footnote omitted).

In this respect, there is *no* requirement that a given methodology be devoid of unprovable inferences or assumptions or that it have no gaps in its reasoning. As *Daubert's* flexible protocol makes clear, some facts are known, some are yet to be known and some may be unknowable.

Second, in fulfilling her *Daubert* obligations, the district judge must also conduct a further inquiry into whether the proposed testimony "is sufficiently tied to the facts of the case that it will aid the jury in resolving the factual dispute," i.e., is "sufficiently relevant to the task at hand." *Daubert*, 509 U.S. at 591; 597. Relevant evidence "means evidence having any tendency to make the existence of any fact that is of consequence to the determination of the action more probable or less probable than it would be without the evidence." Fed. R. Evid. 401. The inquiry is one of "fit," the logical relationship between the proffered proof and the material issue it is supposed to support in order to judge whether it will help the trier of fact. *Id.* at 591, citing *United States v. Downing*, 753 F.2d 1224, 1242 (3rd Cir.1985).

Addressing the concern that this two-step inquiry will lead to juries being confronted with unreliable "quack science" dressed up as scientific analysis, the *Daubert* Court observed:

> [v]igorous cross examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence. See *Rock v. Arkansas*, 483 U.S. 44, 61(1987)....
>
> *These conventional devices, rather than wholesale exclusion under an uncompromising "general acceptance"test, are the appropriate safeguards where the basis of scientific testimony meets the standards of Rule 702.*

Id. at 596(emphasis supplied).

In the instant case, the district judge held three weeks of hearings assessing expert testimony on the etiology of Parkinson's Disease and manganism. As she eventually found, the methodology of Dr. Carlini was neither flawed nor inconsistent with the diagnostic methods used by all the other expert witnesses *for each side* in this case; and it therefore wasn't "junk science," having met most of *Daubert's* indicia of reliability.

By demanding that Dr. Carlini's testimony possess no unprovable inferences or assumptions and that it have no gaps in its reasoning, the majority nullifies *Daubert's* flexible standard for the admission of expert evidence under Rule 702, ignores its liberal standard for the admissibility of such testimony and undercuts the preeminent goal of the Rules of Evidence that claims be "justly determined" and that "the truth may be ascertained." Fed. R. Evid. 102. This result---unraveling three weeks of *Daubert* hearings, eleven days of trial and eight days of jury deliberations----substitutes pure speculation by the court of appeals

about the cause of Tamraz's parkinsonism for the jury's verdict and justifies an order by this Court granting the writ.

The majority dismisses as "speculation" Dr. Carlini's expert opinion as Tamraz's treating neurologist that Tamraz most likely had a genetic predisposition to Parkinson's Disease and that his long-term exposure to manganese as a veteran welder of 25 years working in closely confined spaces without a respirator triggered his underlying predisposition to Parkinson's Disease at the early age of just 44. It criticizes Dr. Carlini's reasoned differential diagnosis for taking too many "speculative jumps" and "leap[s] of faith." In doing so, the majority engages in naked speculation of its own—that treating physicians like Dr. Carlini do not approach etiology determinations with scientific rigor sufficient to pass muster in the courtroom (App. 16-17);

> "Getting the diagnosis right matters greatly to a treating physician, as a bungled diagnosis can lead to unnecessary procedures at best and death at worst....But with etiology, the same physician may often follow a precautionary principle: If a particular factor *might* cause a disease, and the factor is readily avoidable, why not advise the patient to avoid it?....This low threshold for making a decision serves well in the clinic but not in the courtroom, where decision requires not just an educated hunch but at least a preponderance of the evidence."

Id. (emphasis in original).

The majority takes issue with the last four steps of Dr. Carlini's analysis. (App. 9-10). But when examined through the lens of *Daubert's* robust principles of admissibility, each step of Dr. Carlini's analysis demonstrates a practical consistency among the known facts presented to him by Tamraz's condition and history, the relevant literature, Dr. Carlini's own training and experience in treating movement disorders, and those of his peers.

First, the majority dismisses the scientific literature relied upon by Dr. Carlini to form a link between environmental toxins like manganese and a genetic predisposition to Parkinson's Disease as all "theoretical." (App. 10). But, as the dissent observed, this "theoretical" debate in the literature among Dr. Carlini's peers in 2007 was a healthy, evolving one, moving in the direction of recognizing "a large likelihood that what we now know as sporadic Parkinson's disease, which is not understood very well, is due to a combination of environmental factors together with an underlying genetic predisposition." (App. 36). Though not yet "proven" in the scientific sense, this connection was emerging in "a lot of studies" and in "a lot of thinking" by his peers. (App. 36-37).

Indeed, this was more than a mere "hypothesis," as the majority surmised. It aligns with the generally accepted theory, documented in scientific literature since at least 1996, that Parkinson's Disease is thought to develop "as a result of interactions between genetic susceptibility factors and environmental exposures." Sandy et al., *CYP2D6 allelic frequencies in young-onset Parkinson's Disease*, Neurology 47(1):225-230 (1996). Even respondents' own expert at the three-

week *Daubert* hearings, Dr. Warren Olanow, espoused this connection; and their expert at trial, Dr. Anthony Lang, co-authored an article written several months before Dr. Carlini's testimony in 2007, which stated that "[i]t is clear that PD...is *most likely* caused by a combination of genetic and environmental factors." Litvan et al., *The etiopathogenesis of Parkinson's disease and suggestions for future research: Part I*, J. Neuropathol. Exp. Neurol. 66(4):251-257 (2007) (emphasis supplied). None of this adds up to "quack science."

Second, the majority faults Dr. Carlini's analysis for not identifying studies finding a link between manganese and Parkinson's Disease and for admitting that "some" studies which have "looked at that...have not found a very strong correlation." (App. 10). But the studies he was referring to were epidemiological studies of populations living near manganese mines in Norway and Italy that did not show a high rate of *manganism*, a rare disease----*as opposed to* Parkinson's Disease. (J.A. 601-602). How this admission by Dr. Carlini was relevant to his reasoned opinion about the cause of parkinsonism in a welder who was subjected to 25 years of welding fumes containing high concentrations of manganese on a daily basis in close quarters without a respirator is *never* addressed by the majority.

Third, the majority criticizes Dr. Carlini's analysis for "guessing" about Tamraz's underlying predisposition to Parkinson's Disease because Tamraz had no family history of Parkinson's Disease. (App. 10). But Dr. Carlini testified that based upon in his experience and training, "there are probably a large

number of people who have genetic predispositions to parkinsonism out there *who never get it* because their predisposition is just that, not a guarantee....[a]nd unless they have a second hit of some kind like an environmental hit, then they would not develop...Parkinson's disease." (J.A. 601)(emphasis supplied). Accordingly, that Tamraz's family did not have a documented history of Parkinson's Disease does not mean that *he* did not have a predisposition to develop parkinsonism; and thus even if Dr. Carlini could not prove as scientific fact that Tamraz's family had a history of Parkinson's Disease, it would not, as the majority surmised, conclusively defeat his overall theory of causation. (App. 10).

Finally, the majority takes issue with Dr. Carlini's failure to account for the fact that Parkinson's Disease occurs in the general population usually without any identified cause, making it hard to attribute Tamraz's case to manganese over all of the other possible causes, especially when the probability of getting parkinsonism from an underlying predisposition is unknown. (App. 10-12). Yet the majority ignores the core basis for Dr. Carlini's opinion: the convergence of the results of his physical examination and Tamraz's full medical and occupational history, the relevant literature, Dr. Carlini's own training and experience in treating movement disorders and those of his peers.

Employing this standard methodology, which every neurologist in this case used, Dr. Carlini examined Tamraz and took his medical/occupational history, accepted techniques in the neurological community for forming a differential diagnosis. Because Tamraz exhibited classic symptoms closer to

Parkinson's Disease than to those of manganism and because Tamraz experienced "early onset" parkinsonism at the age of just 44, Dr. Carlini believed that Tamraz "most likely has a genetic predisposition to Parkinson's Disease" and that [his long term exposure to] manganese [as a veteran welder subjected to 25 years of welding fumes containing high concentrations of manganese on a daily basis in close quarters without a respirator] tipped him over" into parkinsonism. (J.A. 601;604). He held this diagnosis of manganese-induced or manganese-triggered parkinsonism with a reasonable degree of medical certainty "because that seemed the most likely explanation for [Tamraz's] early onset parkinsonism," a key factor in his diagnosis. (J.A. 601;604;758).

As for Tamraz's early onset, the majority dismisses this crucial factor since ten percent of persons with Parkinson's Disease develop symptoms before age 50, a fact which "does not create an inference that something unusual must have caused it in Tamraz." (App. 11-12). This reasoning usurps the jury's function. This factor mattered very much to Dr. Carlini's probabilistic scientific thinking and it was for the jury to consider its weight since his opinion on this score fell within the "range where experts might reasonably differ." In fact, during the 2005 *Daubert* hearings, respondents' own expert, Dr. Warren Olanow, acknowledged that early onset is a critical, *indeed decisive*, factor in determining the etiology of parkinsonism; and early onset likely indicates a genetic predisposition for the disease. (J.A. 591). Even Dr. Lang, respondents' trial expert, conceded that a genetic form of the disorder could be inferred from Tamraz's early onset of the disease. (J.A. 448).

Moreover, that Dr. Carlini did not perform the most advanced "cutting edge" genetic screening or imaging techniques—*none* of which was available in a clinical setting and one of which could only be performed upon autopsy----to identify the form of Tamraz's parkinsonism does not dilute the force of his opinions. Founded upon a differential diagnosis employing a standard methodology used by every other expert in this case, Dr. Carlini first ruled in manganese, a known neurotoxin, as a cause of Tamraz's disease, given his work history and manganese's potential for causing brain injury; second, he reviewed the scientific literature showing that manganese is capable of causing Tamraz's disease given its capacity to damage all regions of the brain, not just regions associated with manganism, and that "all by itself, [manganese] can cause a form of parkinsonism if it's in sufficient quantity"; and third, he ruled out idiopathic Parkinson's Disease reasoning that based on Tamraz's exposure to manganese for 25 years at his welding job at lower levels than the intoxicating levels which could cause manganism, together with the very early onset of the disease, the manganese exposure was most likely the neurotoxic "hit" that "set off" his genetic susceptibility and tipped him into Parkinson's Disease. See *The Admissibility of Differential Diagnosis Testimony To Prove Causation in Toxic Tort Cases: The Interplay of Adjective and Substantive Law*, 64 Law & Contemp. Probs. 107, 123-124 (2001).

At best, scientists—even experienced treating neurologists like Dr. Carlini—are imperfect eyewitnesses to the scientific enterprise and their opinions should be judged on reasonableness, not by an impossible standard that requires them to rule out with

conclusive finality all other unknown agents or to prove every inference or assumption in their thinking. The majority's insistence that Dr. Carlini do so nullifies *Daubert's* flexible standard for the admission of expert evidence under Rule 702. By definition, "idiopathic" PD is of unidentified etiology and, therefore, is "difficult" to rule out absolutely, as the majority concedes. To exclude Dr. Carlini's opinion for not being able to do so is to hold that all possible alternative causes must be ruled out. This squarely conflicts with decisions rendered by at least two sister circuits, the Third and Fourth. *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 156 (3rd Cir. 1999)("A medical expert's causation conclusion should not be excluded because he or she has failed to rule out every possible cause of the plaintiff's illness."); *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 265 (4th Cir. 1999)(same).

Dr. Carlini used scientific probabilistic reasoning to determine which of two explanations that could not be ruled out absolutely----manganese or idiopathic—was the most likely. *Id.* at 262. That this scientific approach would have sufficed in the Third and Fourth Circuits, but did not in the Sixth, is reason enough to grant the writ, and doubly so due to the majority opinion's conflict with *Daubert* and its progeny. Dr. Carlini's opinion is reliable, relevant proof, founded on fundamentally sound methodology, precisely the type of evidence that *Daubert* expressly anticipated and approved. The district court rightly deemed it appropriate for the jury to hear.

II. The Majority's Use Of A *De Novo* Standard Of Review Instead Of An Abuse of Discretion Standard To Assess The District Judge's Decision To Admit Expert Testimony Usurps The District Judge's "Gatekeeping" Function Under *Daubert* And Undermines *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999) and *General Electric Co. v. Joiner*, 522 U.S.136 (1997).

The more deferential abuse of discretion standard of review is especially necessary in assessing the admissibility of expert testimony under *Daubert* for the reasons identified by the Court in *General Electric Co. v. Joiner*, 522 U.S.136,147-150 (1997) (Breyer, J., concurring), i.e., a stricter standard such as *de novo* review has the potential to undo the sound decisions of the trial judge upon appellate fact-finding having no special worth except as a "second guess" in the aftermath of trial; it undercuts the bias of the Federal Rules of Evidence favoring the admissibility of such evidence; and it denigrates without reason the crucial "gatekeeping" function of the trial court. *Id.* Most important, it "will help secure the basic objectives of the Federal Rules of Evidence, which are...the ascertainment of truth and the just determination of proceedings." *Id.* at 150.

In *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999), the Court reinforced the notion that the trial judge, as *Daubert's* "gatekeeper," be given wide latitude by the appellate court in making her decision to admit or exclude proffered expert testimony and that an abuse of discretion standard of review furthers this goal:

> ...we conclude that the trial judge must have considerable leeway in deciding in a particular case how to go about determining whether particular expert testimony is reliable.
>
> ....Our opinion in *Joiner* makes clear that a court of appeals is to apply an abuse-of-discretion standard when it "review[s] a trial court's decision to admit or exclude expert testimony. 522 U.S. at 138-139. That standard applies as much to the trial court's decisions about how to determine reliability as to its ultimate conclusion....Indeed, the Rules seek to avoid "unjustifiable expense and delay" as part of their search for "truth" and the "jus[t] determin[ation]" of proceedings. Fed. R. Evid. 102. Thus whether *Daubert's* specific factors are, or are not, reasonable measures of reliability in a particular case is a matter that the law grants the trial judge *broad latitude* to determine. See *Joiner*, [522 U.S.] at 143.

526 U.S. at 152-153(emphasis supplied).

As Circuit Judge Wood's dissenting opinion in *Dura Automotive Systems of Indiana v. Cts Corp.*, 285 F.3rd 609, 617 (7th Cir. 2002) makes clear, *de novo* review should be employed, if at all, only to ensure that the district court took all of the steps that a correct application of the *Daubert* framework requires, a purely legal inquiry. *Id.* citing *United States v. Hall*, 93 F.3d 1337, 1341-1342 (7th Cir. 1996). If that legal inquiry is favorable----and it should have been here----- then the court of appeals should have been obligated to review Judge O'Malley's application of the *Daubert* standards under a deferential abuse of discretion

standard. *Id.*, citing *Dhillon v. Crown Controls Corp.*, 269 F.3d 865, 869 (7th Cir. 2001).

The majority's refusal to employ this deferential standard of review undermines not only *Daubert's* robust principles of admissibility but also contravenes this Court's core principles of review enunciated in *Kumho Tire* and *Joiner*, principles deliberately formulated to ensure that the district courts' assiduous attention to the requirements of *Daubert* not be upended by appellate fact-finding which recasts as "quack science" legitimate diagnosis passing most, if not all, of *Daubert's* signposts for admissibility.

CONCLUSION

For all of these reasons identified herein, a writ of certiorari should issue to review the judgment of the United States Court of Appeals for the Sixth Circuit and, ultimately, to vacate and reverse the decision of the court of appeals and remand the matter to the United States District Court for the Northern District of Ohio with instructions to reinstate the jury's verdict against respondents; or provide petitioners with such other relief as is fair and just in the circumstances of this case.

Respectfully submitted,

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Nos. 08-4015/4016
UNITED STATES COURT OF APPEALS
FOR THE SIXTH CIRCUIT

JEFF TAMRAZ; TERRY TAMRAZ, Plaintiffs-Appellees,

v.

LINCOLN ELECTRIC COMPANY; HOBART BROTHERS COMPANY; ESAB GROUP, INC. (08-4015); TDY INDUSTRIES, INC. (08-4016), Defendants-Appellants.

Appeal from the United States District Court for the Northern District of Ohio at Cleveland. No. 04-18948--Kathleen McDonald O'Malley, District Judge.

November 18, 2009, Argued
September 8, 2010, Decided
September 8, 2010, Filed

Before: MARTIN and SUTTON, Circuit Judges; REEVES, District Judge. * SUTTON, J., delivered the opinion of the court, in which REEVES, D. J., joined. MARTIN, J., delivered a separate dissenting opinion.

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OPINION

SUTTON, Circuit Judge. At issue in this case is the often-elusive line between admissible opinion and inadmissible speculation under Rule 702 of the Federal Rules of Evidence and *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 113 S. Ct. 2786, 125 L. Ed. 2d 469 (1993). Several manufacturers of welding supplies appeal a $ 20.5 million jury verdict based on a doctor's testimony that their products triggered "manganese-induced parkinsonism" in a welder who used them. Because the district court exceeded its discretion in allowing this testimony, we reverse and remand for a new trial.

I.

From roughly 1979 to 2004, Jeff Tamraz worked as an

independent-contracting welder in California. Beginning in about 2001, he began to suffer symptoms of Parkinsonism: tremors, drooling, a "masked face" and impaired coordination on his right side. JA 800-03.

In September 2004, Tamraz and his wife Terry sued several manufacturers of welding supplies, alleging that the fumes from their products had caused his condition and that labels on the products had failed to warn of the danger. The case was consolidated with ongoing multidistrict litigation in the Northern District of Ohio. *In re: Welding Fume Prods. Liab. Litig.*, No. 03-cv-17000, MDL No. 1535. The district court selected Tamraz's case for one of several bellwether trials to guide the resolution of the other cases. No. 03-cv-17000, R.2043 (June 6, 2007).

After summary judgment thinned the claims and defendants, Tamraz's case went to trial on three theories of relief (strict-liability failure to warn, negligent failure to warn and fraud by concealment) against five defendants (The Lincoln Electric Company, Hobart Brothers Company, The ESAB Group, Inc., BOC Group, Inc. and TDY Industries, Inc.). The jury found for Tamraz and against all five defendants on the claims of strict liability and negligent failure to warn, but rejected his claim of fraud by concealment. It awarded Jeff Tamraz $ 17.5 million in compensatory damages and Terry Tamraz $ 3 million for loss of consortium. The defendants all filed motions to overturn the verdict under Rule 50 of the Federal Rules of Civil Procedure. The district court denied the challenges of every defendant save BOC Group, against whom the court found insufficient evidence to sustain

the verdict. Lincoln Electric, the ESAB Group, Hobart Brothers and TDY Industries appealed.

II.

The manufacturers argue that the district court should not have admitted Dr. Walter Carlini's opinion that the manufacturers' products triggered "manganese-induced parkinsonism" in Tamraz, claiming it did not satisfy the requirements of Rule 702 of the Federal Rules of Evidence. We agree.

A.

The relevant law. Rule 702 says:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to . . . determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Fed. R. Evid. 702. The rule gives district courts a "gatekeeping role" in screening the reliability of expert testimony, *Daubert*, 509 U.S. at 597, and we review their decisions for abuse of discretion, *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152, 119 S. Ct. 1167, 143 L. Ed. 2d 238 (1999).

The relevant science. Doctors now recognize that what James Parkinson described nearly two centuries ago as "the shaking palsy" makes up a family of movement disorders encompassing Parkinson's Disease along with an assortment of other disorders. James Parkinson, *An Essay on the Shaking Palsy* (1817), *reprinted in* 14 J. Neuropsychiatry & Clin. Neurosci. 223 (2002); *see* JA 130-31. The disorders have different causes, and they have different but overlapping symptoms, including tremors, instability and slowness and rigidity of movement. JA 130. Diagnosing one type of parkinsonism over another is no easy task. JA 140, 553-56.

Two forms of parkinsonism--Parkinson's Disease and manganism--matter here. Parkinson's Disease is the most common type, afflicting more than a million people in the United States alone. JA 131, 607. The typical individual with Parkinson's Disease suffers from a gradual loss of motor function and a tremor when at rest, both usually developing on one side of the body, caused by deterioration of neurons in a part of the brain called (bear with us) the substantia nigra pars compacta. JA 132, 135-36. The causes of Parkinson's Disease range from the obscure to the unknown. As a result, doctors and scientists often define Parkinson's Disease by its undetermined cause--"idiopathic Parkinson's Disease"--"idiopathic" being another way of saying the medical community does not know why a given individual has the disease. In other cases, they use a name other than Parkinson's Disease--such as postencephalitic parkinsonism, drug-induced parkinsonism, or toxin-induced parkinsonism--when they know the cause. JA 131, 150. Over time, as scientists have discovered more genetic and other

causes for Parkinson's Disease, the medical profession has defined more sub-classifications of the disease and has had to rely less frequently on "idiopathic" designations. JA 130-32.

Manganism is a form of parkinsonism defined by its cause: overexposure to manganese, a hard and brittle element that resembles iron but is not magnetic. The symptoms of manganism overlap with Parkinson's Disease but include an action tremor instead of a rest tremor, symmetry of symptoms and a distinct gait ("cock walk"). JA 584, 871, 1002-05. The typical manganism patient suffers neuron deterioration in a different part of the brain from the typical Parkinson's Disease patient--medically speaking, the globus pallidus and the substantia nigra pars reticulata, not the substantia nigra pars compacta--and therapies used to treat Parkinson's Disease often do not work with manganism. JA 134-36, 564-67.

The diagnosis of Jeff Tamraz. Every doctor to examine Jeff Tamraz has reached a different conclusion about where his case fits into this puzzle. No one disputes that he suffers from parkinsonism; the question is what kind and from what cause. The first neurologist to see Tamraz, Dr. Michael Siegel, initially diagnosed Tamraz with "an unusual form of Parkinson's disease due to manganese poisoning," JA 805, but then became less sure about the role of manganese after he consulted medical literature on the subject, JA 808-09. He eventually concluded that Tamraz's condition is closer to Parkinson's Disease than to manganism: Tamraz had a rest tremor, asymmetry of symptoms and no "cock walk." JA 809-11, 816-19. Although he could not rule out manganese as the cause of the illness, JA 812, Dr.

Siegel believed Tamraz's parkinsonism likely resulted from "factors other than exposure to manganese," JA 817.

Tamraz's second neurologist, Dr. Carlini, the witness at issue here, concluded that Tamraz suffers from "manganese-induced parkinsonism," JA 615, but not in the sense of a manifestation of manganism, as that phrase is sometimes used, *see* JA 600-01. He believed that manganese exposure caused something akin to Parkinson's Disease in Tamraz. JA 600. He found many of the same symptoms that led Dr. Siegel to suspect Parkinson's Disease rather than manganism, JA 616, 619-21, but he noted that scientists recently had discovered genetic or environmental causes for many forms of Parkinson's Disease formerly considered idiopathic, JA 599, 619, and discussed literature raising the possibility that genetics and environmental factors may cause a large fraction of Parkinson's Disease cases. JA 599, 601-02. Dr. Carlini hypothesized that Tamraz might have a genetic predisposition to Parkinson's Disease, and that manganese in lower levels than necessary to cause manganism might nevertheless "trigger" the symptoms of Parkinson's Disease, like "the straw that broke the camel's back." JA 598-99. He did not believe Tamraz has Parkinson's Disease in the strict sense--that manganese in his view caused the disease meant by definition it could not be "idiopathic" Parkinson's Disease--but believed it to be otherwise identical to Parkinson's Disease. JA 599-600.

In addition to Tamraz's treating physicians, the plaintiffs and defendants each hired a doctor to examine Tamraz. Dr. Anthony Lang, the defendants' doctor, testified that "Parkinson's disease and manganism . . .

are quite distinct and different," JA 1300, and concluded, based on his examination of Tamraz, that Tamraz has Parkinson's Disease, not manganism, *see* JA 467. Dr. Paul Nausieda, the plaintiffs' doctor, testified that Tamraz does not have Parkinson's Disease but "a manganese-induced movement disorder," essentially manganism. JA 867-68. He relied on the absence of other explanations for Tamraz's parkinsonism and its early onset and, in contrast to the other doctors, found that Tamraz has some symptoms more indicative of manganism than of Parkinson's Disease. *See* JA 866-73.

B.

The manufacturers do not question one aspect of Dr. Carlini's testimony--that Tamraz suffers from a form of parkinsonism. They dispute his conclusion that manganese exposure caused the illness. L.E.C. Br. at 24. To put the distinction in medical terms, they challenge Dr. Carlini's etiology (what caused the disorder diagnosed?), not his diagnosis (what disorder caused the set of symptoms observed?).

The problem here is that, when Dr. Carlini testified that manganese exposure caused Tamraz's condition, he went beyond the boundaries of allowable testimony under Rule 702. In the video-taped deposition played at trial, Dr. Carlini opined that Tamraz has "manganese-induced parkinsonism" "with a reasonable degree of medical certainty." JA 615. But the etiological component of this conclusion--the "manganese-induced" part--was at most a working hypothesis, not admissible scientific "knowledge." Fed. R. Evid. 702. Because the "knowledge" requirement of Rule 702 requires "more

than subjective belief or unsupported speculation," *Daubert*, 509 U.S. at 590, the testimony should have been excluded.

The rest of Dr. Carlini's testimony confirms the speculative nature of this opinion. Under questioning by Tamraz's counsel, Dr. Carlini focused on his diagnosis of parkinsonism and barely explained why he thought manganese caused the disease. He stated only that he diagnosed him with "manganese-induced parkinsonism" because "that seemed the most likely explanation for his early onset parkinsonism," based on his "clinical examination," "Mr. Tamraz's history" "[a]nd just general experience and knowledge about movement disorders." JA 615. Questioning by the manufacturers' counsel brought to the surface his line of reasoning: (1) Tamraz was exposed to welding fumes presumably containing manganese, JA 613; (2) he developed the symptoms of Parkinson's Disease (though not those of manganism), JA 604, 613-14, 616; (3) scientists have identified genetic factors that cause some forms of otherwise "idiopathic" Parkinson's Disease, JA 599; (4) some literature has hypothesized that toxins combined with genetics may cause other cases of Parkinson's Disease, JA 599, 601; (5) manganese is known to cause manganism, so it would be a possible candidate for triggering Parkinson's Disease as well, JA 601; (6) Tamraz may have the genes for Parkinson's Disease, JA 621; and (7) manganese may have triggered these genes and given Tamraz parkinsonism, JA 615.

That is a plausible hypothesis. It may even be right. But it is no more than a hypothesis, and it thus is not "knowledge," nor is it "based upon sufficient facts or

data" or the "product of reliable principles and methods . . . applied . . . reliably to the facts of the case." Fed. R. Evid. 702.

Dr. Carlini acknowledged the speculative jumps involved in steps 4, 5 and 6 of this chain of causation--the steps necessary to his theory that manganese exposure may cause Parkinson's Disease in general. At step 4, he described the literature hypothesizing a link between environmental toxins and latent genetic Parkinson's Disease as "all theoretical." JA 599; *see also* JA 621 ("theoretical writing"). At step 5, he conceded he knew of no studies finding a link between manganese and Parkinson's Disease and that "studies that have looked at that . . . have not found a very strong correlation." JA 602; *see* JA 597, 599, 605, 621; *see also* JA 623 ("Epidemiological studies have failed to find a correlation between manganese and Parkinson's disease."). At step 6, he conceded that "speculation" led him to guess that Tamraz had "an underlying predisposition to Parkinson's disease," JA 621, even though Tamraz has no family history of Parkinson's Disease, JA 613. A negative answer at any one of these steps would defeat his overall theory of causation. The reality that all of them were speculative makes the theory speculative three times over. *Cf. Siharath v. Sandoz Pharm. Corp.*, 131 F. Supp. 2d 1347, 1371 (N.D. Ga. 2001) ("Three scientifically unwarranted 'leaps of faith' exist in this causal chain."), *aff'd*, 295 F.3d 1194 (11th Cir. 2002).

The final step required a leap of faith as well, even ignoring the jumps required to get there. That manganese *could cause* Parkinson's Disease in someone like Tamraz does not show that manganese *did cause*

Tamraz's Parkinson's Disease. Parkinson's Disease occurs commonly in the general population and usually without any known cause. Any given case of Parkinson's Disease thus might have occurred regardless of the manganese exposure, making it hard to attribute one case to manganese over all of the other possible causes. *See Bland v. Verizon Wireless, (VAW) L.L.C.*, 538 F.3d 893, 897 (8th Cir. 2008); *In re Breast Implant Litig.*, 11 F. Supp. 2d 1217, 1228-39 (D. Colo. 1998). This attribution is harder still if, as Dr. Carlini hypothesized, Tamraz already had a genetic predisposition toward it, and even more so if, as Dr. Carlini also acknowledged, the base probability of getting parkinsonism from such a predisposition is unknown. *See* JA 601 (acknowledging that he did not know whether someone with a genetic predisposition toward Parkinson's Disease, as he suspected Tamraz had, would have a 90% chance or a 10% chance of manifesting symptoms).

Dr. Carlini never explained how he made this leap--how *this* case stemmed from manganese exposure. When asked how to "tell the difference between a welder with idiopathic Parkinson's disease and a welder . . . tipped into the Parkinson's disease by welding," he answered with tests he *might* do, not tests he had done. JA 602. Asked similar questions twice more, he responded twice more by listing tests he could do, not tests he had done. *See* JA 603 ("[Y]ou would suspect . . . subtle differences . . . which I bet would be possible to tease out if we had . . . some of these more advanced imaging techniques."); JA 622 ("[W]e haven't tried yet. . . . Suppose--let's just take a hypothetical. . . ."). The closest he came to explaining why he suspected that manganese exposure caused Tamraz's parkinsonism is

when he noted its "early onset." JA 604, 615. But he also said that roughly ten percent of people with Parkinson's develop symptoms before age 50, JA 607, which, considering the high prevalence of Parkinson's Disease, does not create an inference that something particularly unusual must have caused it in Tamraz, who was 41 to 44 years old at the onset of his symptoms, JA 467. (Other witnesses suggested that the frequency of early onset is less than ten percent, *see, e.g.*, JA 573, but that has no relevance to *Dr. Carlini's* methodology.) Dr. Carlini's testimony thus suffers from a lack of foundation both for why manganese could cause Parkinson's Disease and why manganism caused this case of Parkinson's Disease.

Under these circumstances, it makes no difference that Dr. Carlini purported to find "manganese-induced parkinsonism" in Tamraz "with a reasonable degree of medical certainty." JA 615. Whatever Dr. Carlini understood by "with a reasonable degree of medical certainty," the phrase--the conclusion by itself--does not make a causation opinion admissible. The "*ipse dixit* of the expert" alone is not sufficient to permit the admission of an opinion. *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146, 118 S. Ct. 512, 139 L. Ed. 2d 508 (1997). Minus that one phrase, nothing in his testimony suggests the sort of "knowledge" on this point that the Rules require--only speculation, which is generally inadmissible. "[N]o matter how good" experts' "credentials" may be, they are "not permitted to speculate." *Goebel v. Denver & Rio Grande W. R.R. Co.*, 215 F.3d 1083, 1088 (10th Cir. 2000). Dr. Carlini may be a "distinguished" doctor, and "his conjecture" about causation may be "worthy of careful attention . . . . But the courtroom is not the place for scientific guesswork,

even of the inspired sort." *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996).

Rule 702, we recognize, does not require anything approaching absolute certainty. *See Daubert*, 509 U.S. at 590. And where one person sees speculation, we acknowledge, another may see knowledge, which is why the district court enjoys broad discretion over where to draw the line. *See Joiner*, 522 U.S. at 139. Yet, so long as there is a line, some forms of testimony may cross it, and that happened here. Dr. Carlini's opinion contains not just one speculation but a string of them: A suggests by analogy the possibility of B, which might also apply to C, which, if we speculate about D, could eventually trigger E, so perhaps that happened here. At some point, the train becomes too long to pull and the couplings too weak to hold the cars together.

C.

Tamraz resists this conclusion on a number of grounds, all unconvincing. He first turns to Dr. Carlini's use of the phrase "manganese-induced parkinsonism" to describe Tamraz's condition, JA 615, suggesting that Dr. Carlini equates the phrase with manganism, Tamraz Br. at 33 & n.7. If manganism, as its name implies, is caused by manganese, and if Dr. Carlini diagnosed Tamraz with manganism, the thinking goes, the required link between manganese exposure and the disease caused by it has been solved.

The problem is that this argument mischaracterizes Dr. Carlini's testimony. Although some people use "manganese-induced parkinsonism" to refer to manganism, *see, e.g.*, JA 862, Dr. Carlini did not. He

used the phrase to mean Parkinson's Disease that happens to have manganese exposure as its cause:

> Q So you use the term manganese-induced parkinsonism to mean the same disease as Parkinson's disease?
>
> A It's the same disease, yes. It's just triggered by manganese. . . . It's a very specific form of the same disease in the sense that I'm implying that it's triggered by a certain environmental toxin as opposed to another.
>
> Q But it's pathologically and clinically Parkinson's disease?
>
> A It's different, yes, than manganism. Exactly. Pathologically it might look exactly the same as sporadic idiopathic Parkinson's disease, yes.

JA 600. "[E]very aspect" of Tamraz's condition, he added, "is consistent with a diagnosis of Parkinson's disease." JA 607. He used a different name for the disease because idiopathic Parkinson's Disease by definition has no cause, so once he assigned a cause to the disease Dr. Carlini also had to assign a name to that "specific form of the same disease": "manganese-induced parkinsonism." JA 600. But the naming did not change the underlying diagnosis. He repeatedly emphasized that he saw none of the symptoms of manganism in Tamraz and that his diagnosis was identical to Parkinson's Disease save for surmise about its cause. *See* JA 599-600, 616; *see also* JA 601 ("He looks like [idiopathic Parkinson's Disease] clinically."); JA 618 ("Tamraz does not have manganism."). Tamraz's expert,

Dr. Nausieda, who used "manganese-induced parkinsonism" nearly synonymously with manganism, acknowledged that Dr. Carlini did not mean the same thing. JA 862.

In denying the manufacturers' motion to exclude Dr. Carlini's causation testimony, the district court noted that the manufacturers "ask[ed] the Court to draw bright lines regarding diagnoses of movement disorders [i.e., between Parkinson's Disease and manganism] that I have already declined to draw." JA 170-71. But it was not just the manufacturers drawing these lines; Dr. Carlini himself called manganism and Parkinson's Disease "very distinctive" diseases and found Tamraz's symptoms lined up with Parkinson's Disease, not manganism. JA 607. Dr. Carlini's opinion cannot escape its own logic.

In conflating "manganese-induced parkinsonism" with manganism, Tamraz conflates diagnosis with etiology, eliding the distinction between Tamraz's disease and what caused it. Diagnosis *and* etiology, however, both were in play in this case. Because Dr. Carlini diagnosed Tamraz with something akin to Parkinson's Disease, not manganism, and because Parkinson's Disease unlike manganism has no standard etiology, Dr. Carlini's etiology must rise or fall on its own.

To use an analogy, chronic shortness of breath may be caused by diseases ranging from emphysema to lung fibrosis to bronchitis to heart disease--which would be the diagnosis. Heart disease, to pick one of these diagnoses, may be caused by diet, smoking, genetics or some combination of the three--which would be the etiology. One could not defend a verdict without linking

the etiology to the diagnosis. *Cf. Kelley v. Am. Heyer-Schulte Corp.*, 957 F. Supp. 873, 882 (W.D. Tex. 1997) ("Essentially, this is a bit like saying that if a person has a scratchy throat, runny nose, and a nasty cough, that person has a cold; if, on the other hand, that person has a scratchy throat, runny nose, nasty cough, and wears a watch, they have a watch-induced cold.").

Tamraz likewise conflates a doctor's expertise in diagnosis with a doctor's expertise in etiology, arguing for the reliability of Dr. Carlini's causation testimony because of his "extensive . . . experience" with diagnosing parkinsonism. Appellees' Response to Rule 28(j) Letter (Nov. 9, 2009). But most treating physicians have more training in and experience with diagnosis than etiology. *See* David L. Faigman, *Judges as "Amateur Scientists"*, 86 B.U. L. Rev. 1207, 1221-22 (2006); Edward J. Imwinkelried, *The Admissibility and Legal Sufficiency of Testimony About Differential Diagnosis (Etiology)*, 56 Baylor L. Rev. 391, 405 (2004); Mary Sue Henefin et al., Reference Guide on Medical Testimony, in *Reference Manual on Scientific Evidence* 439, 471-72 (2d ed. 2000). When physicians think about etiology in a clinical setting, moreover, they may think about it in a different way from the way judges and juries think about it in a courtroom. *See Siharath*, 131 F. Supp. 2d at 1371-73. Getting the diagnosis right matters greatly to a treating physician, as a bungled diagnosis can lead to unnecessary procedures at best and death at worst. *See Bowers v. Norfolk S. Corp.*, 537 F. Supp. 2d 1343, 1361 (M.D. Ga. 2007). But with etiology, the same physician may often follow a precautionary principle: If a particular factor *might* cause a disease, and the factor is readily avoidable, why not advise the patient to avoid it? Such advice--telling a

welder, say, to use a respirator--can do little harm, and might do a lot of good. *See* Joe G. Hollingsworth & Eric G. Lasker, *The Case Against Differential Diagnosis*: Daubert, *Medical Causation Testimony, and the Scientific Method*, 37 J. Health L. 85, 98 (2004). This low threshold for making a decision serves well in the clinic but not in the courtroom, where decision requires not just an educated hunch but at least a preponderance of the evidence.

None of this means that physicians may not testify to etiology--we have reversed courts for not allowing such testimony, *see, e.g.*, *Hardyman v. Norfolk & W. Ry. Co.*, 243 F.3d 255, 260-67 (6th Cir. 2001)--only that courts must apply the *Daubert* principles carefully in considering it. "The ability to diagnose medical conditions is not remotely the same . . . as the ability to deduce . . . in a scientifically reliable manner, the causes of those medical conditions." *Gass v. Marriott Hotel Servs., Inc.*, 501 F. Supp. 2d 1011, 1019 (W.D. Mich. 2007), *rev'd on other grounds*, 558 F.3d 419 (6th Cir. 2009). Doctors thus may testify to both, but the reliability of one does not guarantee the reliability of the other.

Nor can Dr. Carlini's testimony be defended as a permissibly admissible "differential diagnosis." A differential diagnosis seeks to identify the disease causing a patient's symptoms by ruling in all possible diseases and ruling out alternative diseases until (if all goes well) one arrives at the most likely cause. *See Hardyman*, 243 F.3d at 260-61. We have accepted this kind of testimony before. *See Glaser v. Thompson Med. Co.*, 32 F.3d 969, 977 (6th Cir. 1994).

The manufacturers, however, do not challenge Dr. Carlini's differential diagnosis, which concluded that Tamraz suffers from parkinsonism similar to classical Parkinson's Disease; they challenge his etiology that manganese caused it. Many courts, including our own, allow experts to employ a rule-in/rule-out reasoning process for etiology as well as diagnosis—essentially, a "differential etiology," though the term seems to be a legal one rather than a medical one. *See McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1252 (11th Cir. 2005); Henefin et al., *supra*, at 444, 481. This court's opinions have used "differential diagnosis" broadly to include what might better be called "differential etiology," but they have not had to distinguish the two concepts because most cases involve just one of them. *See, e.g., Hardyman*, 243 F.3d at 259 n.2 (parties did not dispute the nature of the disease, only what caused it).

Whether we describe Dr. Carlini's causation methodology as "differential etiology" or "differential diagnosis," that does not make it reliable. "[S]imply claiming that an expert used the 'differential diagnosis' method is not some incantation that opens the *Daubert* gate." *Bowers*, 537 F. Supp. 2d at 1360. Calling something a "differential diagnosis" or "differential etiology" does not by itself answer the reliability question but prompts three more: (1) Did the expert make an accurate diagnosis of the nature of the disease? (2) Did the expert reliably rule in the possible causes of it? (3) Did the expert reliably rule out the rejected causes? If the court answers "no" to any of these questions, the court must exclude the ultimate conclusion reached. *See Best v. Lowe's Home Ctrs., Inc.*, 563 F.3d 171, 179 (6th Cir. 2009). Dr. Carlini's opinion fails the last two prongs because, for the reasons

already given, his efforts to "rule in" manganese exposure as a possible cause or to "rule out" other possible causes turned on speculation, not a valid methodology. No matter the label, the testimony does not satisfy Rule 702.

All of this suffices to distinguish the cases on which Tamraz relies to admit "differential diagnosis" testimony. In *Hardyman*, the trial court excluded a doctor's opinion that a railroad brakeman's job activities caused his carpal tunnel syndrome (CTS), finding the testimony unreliable because, although the doctor showed that tasks like those the brakeman performed are known to cause CTS, he cited no studies performed on brakemen and could not quantify how much movement and pressure would lead to how much CTS. 243 F.3d at 261-65. We reversed, holding that the district court demanded too much specificity and too much quantification from the expert. *Id.* at 262, 265. But here the problem is not that Dr. Carlini failed to cite studies about manganese causing Parkinson's Disease in welders or could not quantify how much manganese would lead to how much Parkinson's Disease; the problem is that he failed to cite *any* non-speculative evidence for his conclusion that manganese causes Parkinson's Disease.

Likewise, in *Best*, we reversed the district court for excluding a doctor's testimony that a chemical spill on Best's face caused him to lose his sense of smell. 563 F.3d at 183-84. In that case, Best suffered burns on his skin and irritation to his nasal passages immediately after the incident, and eventually lost his ability to smell altogether. *Id.* at 174. We approved the doctor's method of ruling in the chemical in question by making

a careful comparison with similar chemicals he had known to have the same effect. Id. at 181. In this case, by contrast, Dr. Carlini analogized only to the "theoretical" possibility of other toxins causing Parkinson's Disease, JA 599, and did not point to any similar elements known to cause Parkinson's Disease. (He did mention one chemical known to cause parkinsonism--the designer drug MPTP--but never attempted to compare it with manganism and did not factor it into his etiology, JA 619.) In *Best*, the doctor also reliably ruled out most alternative causes; the defendant argued that the doctor also should have ruled out another possible factor but did not provide any evidence that this factor could cause the disease. 563 F.3d at 181. Here, though, the other possibility--unknown (idiopathic) causation--currently accounts for the vast majority of Parkinson's Disease cases, making it impossible to ignore and difficult to rule out. *See Bland*, 538 F.3d at 897. Forecasting today's decision, *Best* cautioned that "[n]ot every opinion that is reached via a differential-diagnosis method will meet the standard of reliability required by *Daubert*." 563 F.3d at 179.

Tamraz invokes the testimony of Dr. Nausieda, who also testified that manganese exposure caused Tamraz's sickness with no objection from the manufacturers. If Dr. Nausieda's testimony passes muster, Tamraz claims, so too should Dr. Carlini's. Tamraz Br. at 35. But we are aware of no authority, and Tamraz points to none, holding that preserving an objection to one witness requires objecting to every similar witness. Although Dr. Nausieda's testimony in some ways overlapped with Dr. Carlini's, moreover, he reached opposite conclusions on the relevant points: He believed

that Tamraz suffers from something akin to manganism, not Parkinson's Disease, JA 866-73, and he believed manganese exposure could not cause Parkinson's Disease. JA 951. Perhaps most importantly, even to the extent that Dr. Nausieda's testimony was consistent with parts of Dr. Carlini's, that would not make Dr. Carlini's testimony admissible. The important thing is not that experts reach the right conclusion, but that they reach it via a sound methodology. *See Daubert*, 509 U.S. at 595. Comparisons between methodologies no doubt may be instructive, and an expert may in some circumstances rely on other experts' testimony, *see* Fed. R. Evid. 703--something Dr. Carlini did not do here. But testimony still must be judged by its methodology, not its conclusion.

Dr. Carlini's speculation that Tamraz might have damage to the globus pallidus in his brain--noteworthy because globus pallidus damage characterizes manganism, not Parkinson's Disease, Tamraz Br. at 33 & n.7--is beside the point. Dr. Carlini primarily expected to see cell deterioration in the substantia nigra pars compacta, which Parkinson's Disease characteristically damages. JA 601. That Dr. Carlini guessed Tamraz *also* might have damage in the globus pallidus--and it was nothing more than a guess, *see* JA 601 ("this is all highly speculative obviously")--neither undermines his diagnosis nor supports his etiology. Globus pallidus damage would be relevant only if Dr. Carlini had actually *detected* globus pallidus damage. That, however, was not the case. There is "too great an analytical gap between the data and the opinion proffered" for the court to admit Dr. Carlini's opinion as testimony. *Joiner*, 522 U.S. at 146.

D.

Having concluded that Dr. Carlini's causation testimony exceeded the permissible boundaries of Rule 702, we must reverse unless we can "say, with fair assurance, . . . that the judgment was not substantially swayed by the error." *Mike's Train House, Inc. v. Lionel, L.L.C.*, 472 F.3d 398, 409-10 (6th Cir. 2006). The error was not harmless.

The emphasis Tamraz put on Dr. Carlini's testimony confirmed its importance. His counsel argued to the jury that "Dr. Carlini alone proves by a preponderance of the evidence that this man has managnese-induced parkinsonism." JA 1407. In their opening argument, their closing argument and again in their rebuttal to the manufacturers' closing argument, counsel for Tamraz played the portion of the video deposition in which Dr. Carlini stated that Tamraz has "[m]anganese-induced parkinsonism" and stated that he held that belief "with a reasonable degree of medical certainty." JA 1317, 1374, 1395. Tamraz's attorney emphasized this supposed certainty: "They are going to tell you he is talking about speculation and conjecture. He is talking about a reasonable degree of medical certainty. . . . That is the standard, and he believes in it." JA 1374-75.

Counsel for Tamraz also leaned heavily on Dr. Carlini's apparent neutrality, calling him a man who "doesn't have a dog in this hunt," JA 1397, and repeatedly mentioning that Dr. Carlini received no payment for his testimony, unlike the manufacturers' only expert, *see* JA 1317, 1374-75, 1399-1400, 1411. They also emphasized Dr. Carlini's treating role: "regardless of what is decided in this case, Jeff Tamraz is going to fly

back home, and next month he is going to walk into Dr. Carlini's office for his December scheduled appointment, and they are going to sit down and talk about how to handle his manganese-induced Parkinsonism. That's the diagnosis." JA 1400-01.

Tamraz argues that Dr. Carlini's testimony could not be harmful because Dr. Nausieda also testified that Tamraz had "manganese-induced parkinsonism," so the jury would have heard the same conclusion even without Dr. Carlini's testimony. But, as shown, Dr. Nausieda and Dr. Carlini meant different things by the phrase "manganese-induced parkinsonism." *Compare* JA 600, *with* JA 862. Dr. Carlini was the only expert who testified that Tamraz had the equivalent of Parkinson's Disease caused by manganese. Without Dr. Carlini, Tamraz would have had to convince the jury that Tamraz suffered from manganism, not Parkinson's Disease. With Dr. Carlini's testimony, however, the jury faced three choices, two of which helped Tamraz: Tamraz won if he had manganism (as Dr. Nausieda testified) or Parkinson's Disease caused by manganese (as Dr. Carlini testified); the manufacturers won only if Tamraz had Parkinson's Disease not caused by manganese. Dr. Carlini's testimony thus shifted the primary question from what disease Tamraz had to whether manganese caused it.

The plaintiffs' closing argument accordingly played down the differences between manganism and Parkinson's Disease and played up the causation issue, noting that three out of four neurologists who had examined Tamraz opined one way or another that manganese had caused Tamraz's illness, which "could be a textbook example in law school of preponderance of

the evidence. There is more evidence in front of you that his disease was caused by manganese in welding fumes than [that it wasn't]. That is our burden." JA 1407-08; *see also* JA 1375 ("The only neurologist of the four that is telling you that 20-plus years of inhaling manganese . . . has nothing to do with his condition is the one they hired and the one that they paid."). Given the importance of Dr. Carlini's testimony, we cannot say "with fair assurance" that the result would have been the same without it.

Our ruling, however, is a narrow one. The manufacturers here do not challenge the district court's primary *Daubert* ruling on Parkinson's Disease testimony, *In re Welding Fume Prods. Liab. Litig.*, No. 03-cv-17000, 2005 U.S. Dist. LEXIS 46164, 2005 WL 1868046, at *22-37 (N.D. Ohio Aug. 8, 2005), and so we do not decide whether other experts may testify that manganese exposure causes Parkinson's Disease. We simply hold that the causation analysis in Dr. Carlini's deposition fell short of what *Daubert* requires.

We leave it to the able district court judge on remand presiding over this difficult case to decide whether to (1) present Dr. Carlini's deposition minus his attribution of Tamraz's illness to manganese (as the manufacturers requested below, JA 366); (2) exclude Dr. Carlini's depositions altogether if the court determines it cannot or should not sever his purely diagnostic conclusions from his etiological hypothesis (as Tamraz suggested, in the alternative, below, JA 461); (3) allow the parties to redepose Dr. Carlini; or (4) opt for any other amenable solution. All we conclude is that his testimony should not have been admitted as it was.

* * * *

No one should construe this opinion as criticism of Dr. Carlini, whom the deposition shows to be intelligent and knowledgeable about the subject matter--immeasurably more so than we are. But not everything a knowledgeable person says is "knowledge" under Rule 702, no more than everything a scientist says is "scientific." "[A] district court judge asked to admit scientific evidence must determine whether the evidence is genuinely scientific, as distinct from being . . . speculation offered by a genuine scientist." *Rosen*, 78 F.3d at 318. This causation opinion fell into the latter category and therefore should have been excluded.

The sort of hypothesis Dr. Carlini presented can play a valuable role both in medicine, where, if the costs of action are low, doctors may want to act on hypotheses without further support, and in science generally, where all discoveries start as untested hypotheses. From this perspective, criticizing Dr. Carlini's hypothesis for being speculative would be like criticizing a sapling for being short. Some hypotheses become scientific theories and others do not.

But that is not the issue. The issue is the reliability of his opinion from a *legal* perspective. And what science treats as a useful but untested hypothesis the law should generally treat as inadmissible speculation. As the Supreme Court has explained, "[t]he scientific project is advanced by broad and wide-ranging considerations of a multitude of hypotheses, for those that are incorrect will eventually be shown to be so . . . . Conjectures . . . are of little use, however, in the project of reaching a quick, final, and binding legal judgment--

often of great consequence--about a particular set of events in the past." *Daubert*, 509 U.S at 597. "Law lags science; it does not lead it." *Rosen*, 78 F.3d at 319.

This is an imperfect system, to be sure. Both sides agree that Mr. Tamraz is a good man who suffers from a terrible disease; we now force him to take the chance of prevailing at trial a second time, with less evidence than before. If he does not, yet it turns out ten years from now that manganese causes his disease, that result will seem unfair. But the alternative route--allowing the law to get ahead of science--would be just as unfair. Such an approach would destroy jobs and stifle innovation unnecessarily. *See Joiner*, 522 U.S. at 148-49 (Breyer, J., concurring); *see also, e.g.*, Gina Kolata, *Panel Confirms No Major Illness Tied to Implants*, N.Y. Times, June 21, 1999, at A1 (describing how scientists concluded, after years of litigation, billions in settlements and the bankruptcy of a major manufacturer, that no evidence tied breast implants to health problems). Rule 702 at all events has drawn the line for us, and we must enforce it. *See Daubert*, 509 U.S. at 597. Because this testimony crossed that line, we reverse.

III.

In view of our decision to vacate the jury verdict, we need not reach the other arguments raised on appeal. We note, however, that the contours of the parties's dispute about the propriety of a "sophisticated user" jury instruction have changed in view of intervening law. After trial, the California Supreme Court endorsed and clarified the defense, *see Johnson v. Am. Standard, Inc.*, 43 Cal. 4th 56, 74 Cal. Rptr. 3d 108, 179 P.3d 905

(Cal. 2008), and as a result the district court may wish to consider this new authority in determining the propriety of such an instruction at a new trial.

IV.

For these reasons, we reverse and remand for a new trial.

Footnote

*The Honorable Danny C. Reeves, United States District Judge for the Eastern District of Kentucky, sitting by designation.

DISSENT

BOYCE F. MARTIN, JR., Circuit Judge, dissenting. The majority finds that the district court abused its discretion in admitting Dr. Walter Carlini's testimony because it "went beyond the boundaries of allowable testimony under rule 702" (*ante* at 6), because it was "speculative" (*ante* at 6), and because his deductions required "leaps of faith." (*Ante* at 8.) Because the majority reached this conclusion by acting as sitting judges rather than under the proper standard of review, I respectfully dissent.

I.

Jeff Tamraz's case is part of a larger multi-district litigation regarding inhalation of manganese fumes by welders. *In re Welding Rod Prods. Liab. Litig.*, 269 F. Supp. 2d 1365 (J.P.M.L. 2003). In April, May, and June 2005, the multi-district litigation court conducted three

weeks of *Daubert* hearings to test the methodologies of expert witnesses. As part of the hearing, experts in the neurological community testified regarding the connection between manganese exposure and various forms of parkinsonism. The court also heard argument on the defendants' motion to preclude evidence that manganese exposure causes Parkinson's Disease. The trial court concluded that the evidence proffered was "sufficiently reliable to support the assertion that exposure to welding fumes can cause, contribute to, or acclerate a parkinsonian syndrome that some doctors can diagnose as [Parkinson's Disease] . . . at least in the abstract, as the question is presented here." (Corrected J.A. at 166, Order, Aug. 6, 2005).

Jeff Tamraz worked as a welder from 1979 to 2004. Around 2000-2001, he began experiencing severe neurological symptoms, which eventually became so severe that he could not care for himself. In July 2007, Tamraz brought suit in the United States District Court for the Northern District of Ohio against the five defendants who provided the welding materials that Tamraz used during his welding career. He claimed that his neurological injuries, which manifested symptoms consistent with Parkinson's Disease, were caused by manganese exposure.

Before trial, the defendants moved to exclude parts of the testimony to be presented by one expert, Dr. Carlini. On November 1, 2007, the court ruled on the motion, stating:

> I have read all of the briefs. I have read . . . the two depositions that were taken of Dr. Carlini. I have gone back and reread the Court's *Daubert*

> opinion, which was on the main MDL docket . . . and I have decided that I am going to deny the defendants' motion.
>
> To a large extent, the defendants' motion asks the Court to draw bright lines regarding diagnoses of movement disorders that I have already declined to draw, and I have already decided that the current state of the science does not require to be drawn. . . . I see nothing about [Dr. Carlini's] methodology that is either flawed or inconsistent with the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used and have described as appropriate diagnostic methods. . . . It is clear that the defendants have fair grounds to attack the somewhat unusual diagnosis that Dr. Carlini renders in this case . . . but that to me goes to the weight and not the admissibility of his testimony.

(Corrected J.A. at 170-173, Tr. of Proceedings, Nov. 1, 2007). At trial, four experts testified regarding the cause of Tamraz's injury. Tamraz's primary medical expert, Dr. Paul Nausieda, testified that Tamraz suffered from manganese-induced parkinsonism. The defendants' lead medical expert, Dr. Anthony Lang, while testifying that manganese exposure can cause parkinsonism, stated that he did not believe that Tamraz's parkinsonism was caused by manganese exposure. Tamraz's former treating neurologist, Dr. Michael Siegel, first diagnosed Tamraz with an unusual form of Parkinson's Disease due to manganese poisoning. He later revised his opinion to state that, while he could not rule out the possibility than

manganese exposure caused Tamraz's injury, Tamraz likely suffered from parkinsonism resulting from factors other than manganese exposure. Finally, Dr. Carlini testified that Tamraz likely had a genetic predisposition to Parkinson's Disease and that exposure to manganese triggered his Parkinson's to develop. The jury found defendants liable and awarded Tamraz a total of $ 20.3 million in compensatory damages.

Defendants appealed, claiming that the district court erred in admitting Dr. Carlini's testimony because it was speculative and not based upon published literature or scientific studies. The majority agrees and reverses the district court's decision. For the reasons that I will discuss below, I respectfully disagree with the majority's reasoning and conclusions.

II.

As the majority correctly notes, "[t]his court reviews a district court's decision concerning expert testimony for abuse of discretion." *Popovich v. Sony Music Entertainment*, 508 F.3d 348, 359 (2007) (quoting *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 152-53, 119 S. Ct. 1167, 143 L. Ed. 2d 238 (1999)). Unfortunately, while paying lip service to the correct standard, the majority actually applies a de novo standard of review. As we have held, "A district court abuses its discretion if it bases its ruling on an erroneous view of the law or a clearly erroneous assessment of the evidence." *Ky. Speedway, LLC v. Nat'l Assoc. of Stock Car Auto Racing, Inc.*, 588 F.3d 908, 915 (6th Cir. 2009) (quoting *Brown v. Raymond Corp.*, 432 F.3d 640, 647 (6th Cir.2005)) (internal

quotation marks omitted). "Thus, we will not substitute our own judgment for that of the district court and will reverse an evidentiary decision 'only where we are left with a definite and firm conviction that [the district court] committed a clear error of judgment.'" *In re Scrap Metal Litigation*, 527 F.3d 517, 528 (6th Cir. 2008) (quoting *Conwood Co., L.P. v. U.S. Tobacco Co.*, 290 F.3d 768, 781 (6th Cir. 2002)); *see also Nolan v. Memphis City Schools*, 589 F.3d 257, 265 (6th Cir. 2009) (holding that "[b]road discretion is given to district courts in determinations of admissibility . . . and those decisions will not be lightly overturned.").

"[Abuse of discretion review] requires a reviewing court to be highly deferential when assessing not just a trial court's analysis of each [*Daubert*] factor, but also the trial court's initial selection of which factors are relevant to the case at hand." *Johnson v. Manitowoc Boom Trucks, Inc.* 484 F.3d 426, 430 (6th Cir. 2007). It is within the district court's discretion to determine whether the testimony provided is inadmissible "junk science" or testimony falling within the "range where experts might reasonably differ." *Kumho*, 526 U.S. at 153. Thus, we must conduct our review of that decision with great deference.

Here, because the district court found that Dr. Carlini's "methodology . . . [was neither] flawed or inconsistent with the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used and have described as appropriate diagnostic methods", (corrected J.A. at 170-173, Tr. of Proceedings, Nov. 1, 2007), it is far from apparent that the district court should have found Dr. Carlini's testimony to be unreliable. While the district court

acknowledged that Dr. Carlini's diagnosis was "unusual", nothing in *Daubert* and its progeny indicates that an unusual diagnosis alone renders a district court's decision to admit an expert's testimony an abuse of discretion. Here, the district court reasonably evaluated Dr. Carlini's testimony in light of a broad range of expert opinions and found that it was sufficiently reliable to be admissible. As it was not a clearly erroneous decision so as to constitute an abuse of discretion, the district court's judgment should be affirmed.

Expert testimony is inherently difficult to evaluate. It is all the more so outside the context of a trial. This is why the application of *Daubert* is flexible. It is why the district court, which is able to hear and evaluate experts first-hand, is given such broad latitude in determining whether testimony is admissible. That same reasoning counsels that this Court interfere only in cases where it is absolutely clear that the testimony is nothing more than "junk science" that the jury cannot be trusted to evaluate. That is not the case here.

In reversing the district court's decision, the majority substitutes their opinion for that of the district court and exercises a standard of review closer to de novo than abuse of discretion. The upshot of the majority's opinion is that they would have found Dr. Carlini's testimony inadmissible had they been the trial judge, which would be acceptable if we reviewed this case de novo. However, the majority does little to explain why the district court's decision to admit Dr. Carlini's testimony was "arbitrary, unjustifiable, or clearly unreasonable", *Plain Dealer Pub. Co. v. City of Lakewood*, 794 F.2d 1139, 1148 (6th Cir. 1986), as they

must do if they wish to reverse the district court's evidentiary conclusions under the abuse of discretion standard. Although they go to great lengths to explain why they are dissatisfied with Dr. Carlini's "thrice-speculative" testimony, they have not shown why admitting expert testimony, which relies on "the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used" is an abuse of discretion. (Corrected J.A. at 170-173, Tr. of Proceedings, Nov. 1, 2007). In my view, the majority has, with long arms and short sight, reached much further than our standard of review permits. For these reasons, I cannot join the majority's opinion.

III.

The majority offers several reasons for reversing the district court's opinion, none of which I find persuasive. They criticize Dr. Carlini's testimony for being "speculative" (*ante* at 6) and for its "leaps of faith." (*Ante* at 8.) They also claim that Dr. Carlini confused etiology with diagnosis. (*Ante* at 11.) They further take issue with the idea of admitting his testimony under a "differential diagnosis" analysis because many incidents of Parkinson's Disease are idiopathic,[1] a cause which, by its very definition, cannot be "ruled out." (*See ante* at 14-15.) I believe that refocusing the question on the underlying issue that *Daubert* and its progeny intended to address--the exclusion of "junk science"--and reviewing the district court's evidentiary decisions through the appropriate abuse of discretion lens, leaves us no choice but to affirm the district court's evidentiary conclusions.

A. Dr. Carlini's Testimony is Admissible under Daubert

The path charting the judiciary's standards for admitting or excluding expert testimony--from the early *Frye* standard to *Kumho's* clarification of *Daubert*--has been a movement towards granting district judges greater discretion in making expert testimony determinations. *See Kumho*, 526 U.S. at 137; *Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 113 S. Ct. 2786, 125 L. Ed. 2d 469 (1993); *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923). Today, that discretion is flexible and very broad. *Kumho*, 526 U.S. at 153.

> *Daubert's* role of 'ensur[ing] that the courtroom door remains closed to junk science,' *Amorgianos v. Nat'l R.R. Passenger Corp.*, 303 F.3d 256, 267 (2d Cir. 2002), is not served by excluding [medical expert] testimony . . . that is supported by extensive relevant experience. Such exclusion is rarely justified in cases involving medical experts as opposed to supposed experts in the area of product liability. *See generally* Daniel W. Shuman, *Expertise in Law, Medicine, and Health Care*, 26 J. Health Pol. Pol'y & L. 267 (2001) (characterizing the effect of the *Daubert* and *Kumho Tire* cases on claims of medical expertise as '[m]uch ado about little,' while noting that these cases have had a significant effect on toxic tort and products liability litigation).

Dickenson v. Cardiac and Thoracic Surgery of E. Tenn., 388 F.3d 976, 982 (6th Cir. 2004). "As

'gatekeeper,' the trial judge is imbued with discretion in determining whether or not a proposed expert's testimony is admissible, based on whether it is both relevant and reliable." *Johnson*, 484 F.3d at 429 (quoting *Kumho*, 526 U.S. at 147). It is for the district court to determine whether expert testimony is essentially "junk science" rather than testimony falling within the "range where experts might reasonably differ." *Kumho*, 526 U.S. at 153.

One way in which a court may make this determination is by examining the expert's testimony in relation to the factors laid out by the Supreme Court.

> These factors include: (1) whether a theory or technique . . . can be (and has been) tested; (2) whether the theory has been subjected to peer review and publication; (3) whether, with respect to a particular technique, there is a high known or potential rate of error and whether there are standards controlling the technique's operation; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community."

Johnson, 484 F.3d at 430 (internal quotations omitted). Six years after issuing *Daubert*, the Supreme Court clarified that "the factors listed [in *Daubert*] do not constitute a 'definitive checklist or test.'" *Id.* at 429-30 (quoting *Kumho*, 526 U.S. at 150). Our Court has "recognized that the *Daubert* factors 'are not dispositive in every case' and should be applied only 'where they are reasonable measures of the reliability of expert testimony.'" *In re Scrap Metal*, 527 F.3d at 529 (quoting *Gross v. Comm'r*, 272 F.3d 333, 339 (6th Cir. 2001)).

"Rather, the gatekeeping inquiry must be tied to the facts of a particular case, depending on the nature of the issue, the expert's particular expertise, and the subject of his testimony." *Id.*

While Dr. Carlini testified that he was not able to point to a specific study showing that manganese exposure caused Parkinson's Disease, his testimony was supported by his own general experience and knowledge (corrected J.A. at 615, Tr. Testimony of Walter Carlini, Sept. 13, 2007), and theoretical medical writing that explored the connection between manganese exposure and Parkinson's Disease.[2] (*Id.* at 599, Tr. Testimony of Walter Carlini, Sept. 11, 2007). When asked what publications substantiated his claim, Dr. Carlini clarified that "[t]here is a lot of literature out there about the potential--and it's all theoretical--about the potential causes for sporadic parkinsonism. And a lot of literature discusses the combination of environmental factors together with genetic predispositions." (*Id.*) He further stated that "there is a large likelihood that what we now know as sporadic Parkinson's disease, which is not understood very well, is due to a combination of environmental factors together with an underlying genetic predisposition. That's the way the field is moving." (*Id.*) He additionally testified that, "there is [sic] a lot of studies or a lot of thinking out there . . . which conceptualizes sporadic Parkinson's disease as being . . . a combination of environmental factors and genetic predisposition which is how I conceive of manganese-triggered parkinsonism that falls under that rubric." (*Id.* at 600.) He further stated that "there is quite a bit of writing about patients--theoretical writing about patients developing Parkinson's disease due to a combination of genetic and

environmental factors." (*Id.* at 621, Tr. Testimony of Walter Carlini, Sept. 13, 2007).

Thus, the connection between manganese and Parkinson's disease, though not agreed upon by every member of the scientific community, was certainly the subject of valid scientific debate and publication at the time of Dr. Carlini's testimony.[3] The district court succinctly explained its decision not to exclude Dr. Carlini's evidence, focusing on his methodology: "I see nothing about [Dr. Carlini's] methodology that is either flawed or inconsistent with the very diagnostic methods that other experts in this case . . . have used and have described as appropriate diagnostic methods." (*Id.* at 170-173, Tr. of Proceedings, Nov. 1, 2007).

While Dr. Carlini's testimony may not have satisfied every *Daubert* factor, it is not necessary that it do so. *Johnson*, 484 F.3d at 429-30 (holding that the factors do not constitute a definitive checklist or test); *see also In re Scrap Metal*, 527 F.3d at 529 (holding that the *Daubert* factors "are not dispositive in every case and should be applied only where they are reasonable measures of the reliability of expert testimony." (internal quotations omitted)). Dr. Carlini's testimony easily satisfied at least one *Daubert* factor because the manganese-Parkinson's Disease theory was the subject of peer review and publication at the time of Dr. Carlini's testimony. *See infra*, n.2.

Furthermore, to the extent that the connection between manganese and Parkinson's Disease could be tested at the time, the then-ongoing studies of individuals exposed to manganese, who later developed Parkinson's Disease, constitutes testing sufficient to

satisfy *Daubert*. Therefore, Dr. Carlini's testimony appears to meet one, if not several, *Daubert* requirements. Thus, the district court did not abuse its discretion in admitting it, and the majority errs in so holding.

B. Speculation and Gaps in the Testimony

The majority finds that Dr. Carlini's testimony was speculative, stating without support that the testimony was "no more than a hypothesis, [and is] thus not 'knowledge,' nor is it 'based upon sufficient facts or data' or the 'product of reliable principles and methods . . . applied . . . reliably to the facts of the case." (*Ante* at 7.) I disagree that Dr. Carlini's testimony was speculative. Based on the record, it seems clear that Dr. Carlini was relying upon scientific studies which tested the causal connection between manganese exposure and Parkinson's Disease. Furthermore, the district court was exercising its broad discretion when it found that Dr. Carlini's methodology was reliable and consistent with the diagnostic methods used by other experts in the case. It seems incredible that the majority--exercising a standard of review that seems closer to de novo than abuse of discretion, and without the benefit of having sat through the hearings and seen the experts--finds Dr. Carlini's testimony to be speculative.

The majority also cites gaps in Dr. Carlini's testimony as a reason to reverse the district court. (*Ante* at 8.) However, the majority's newly-minted requirement that scientific testimony must be without flaws or gaps and have no unprovable inferences or assumptions runs counter to any reasonable understanding of how

scientific "truth" is reached. "Scientists disprove things. In the process they filter error from theories and methodology, but they do not prove that the surviving methodologies--those that are left standing or those that are changed to correct errors--are valid." Jan Beyea & Daniel Berger, *Scientific Misconceptions among* Daubert *Gatekeepers: The Need for Reform of Expert Review Procedures*, 64 LAW & CONTEMP. PROBS. 327, 337 (2001). Furthermore, the "theories that survive testing still have components that have never been tested, contain subjective elements, and require that reasonable inferences be made if they are to be used in real world examples." *Id.* At least one other Circuit court has found that "to the extent that [the defendant] asserts there were gaps or inconsistencies in the reasoning . . . such arguments go to the weight of the evidence, not its admissibility." *Campbell v. Metro. Prop. and Cas. Ins. Co.* 239 F.3d 179, 186 (2d Cir. 2001) (the trial court did not abuse its discretion in admitting expert testimony that plaintiffs were suffering from lead poisoning).

Indeed, the most cherished of scientific "truths" are the subject of constant refinement and are frequently overturned by subsequent science. For instance, the 42-year consensus that DNA alone determines heredity was later "dethroned as a universal principle, albeit after the 1994 article by Black et al. was published." *Id.* at 335. In fact, simultaneously accepted scientific principles are sometimes incompatible, and thus, might fare badly under a strict *Daubert* application.

> Imagine Euclid testifying in a modern day Daubert proceeding: 'Professor Euclid, I understand that one of your postulates is that

> parallel lines do not meet at infinity. Can you prove this to be true? Have you ever tested this? Isn't it also true that Professor Einstein has proven that your geometry doesn't work in the presence of gravity?'

Id. at 335 n. 42.

While the district court must necessarily draw lines, we must use caution in demanding the type of finality from science that we have come to expect in law. This is especially true when considering cases of newer scientific studies. It seems to me an overly harsh test at the admissibility level to insist upon testimony with no "gaps", when the science itself may be incapable of absolute proof. Do malfeasing defendants get a free pass on the first few victims because there is not yet a sufficient sample set to create scientific studies with no discernable gaps? Do we tell the early victims, "I'm sorry, you had the misfortune of getting sick too soon", and send them home?

The fact that scientists have not reached consensus regarding medical causation does not render reliance on a scientist's theory improper expert testimony, particularly when, as in this case, the expert is relying on studies that appear to have been conducted using standard methodology. Rather, those differences should go to the weight that a jury should give an expert's testimony. *See Best v. Lowe's Home Centers, Inc.*, 563 F.3d 171, 182 (6th Cir. 2009) (finding that "admissibility under 702 does not require perfect methodology. . . . Any weakness in [a "competent, intellectually rigorous physician's"] methodology ["in identifying the most likely cause of the plaintiff's injury"] will affect the

weight that his opinion is given at trial, but not its threshold admissibility."). In cases where the state of scientific consensus is difficult to determine, we must defer to the district court. The district court has the distinct advantage of having heard all the experts testify and can weigh the reliability of a given expert's testimony against others more easily than we can. Our valuations of complicated medical expert issues such as these are made out of context and are therefore more likely to suffer flaws.

Because Dr. Carlini relied on scientific methodology used by other experts in his field, *see infra* at 28, I do not believe that the district court abused its discretion in admitting his testimony. The district court's determination that Dr. Carlini's methodology was sufficiently reliable was certainly not clearly erroneous, so the testimony was admissible. What weight to grant his testimony was a question for the jury, not an appellate court sitting far removed from the trial. Because the majority has not demonstrated that the district court abused its discretion, it errs in reversing the district court's decision.

IV.

For the reasons above, I respectfully dissent.

Footnotes

1Idiopathic disease are those for which there is no known cause--although Dr. Gregory House may provide the better definition: "Idiopathic, from the Latin meaning we're idiots 'cause we can't figure out what's

causing it." *House: Role Model* (Fox television broadcast Apr. 12, 2005).

2We must evaluate Dr. Carlini's testimony in light of the science available to him at the time. Any findings--positive or negative--regarding the causal connection between manganese and Parkinson's disease made since that time are irrelevant for this analysis. It is clear that he was referring to a then-ongoing debate regarding the causal connection between manganese exposure and Parkinson's Disease. *See* Murry M. Finkelstein, Michael Jerett, *A Study of the Relationships between Parkinson's Disease and Markers of Traffic-Derived and Environmental Manganese Air Pollution in Two Canadian Cities*, 104 ENVTL.RES. 420-432 (2007); *Link Found Between Parkinson's Disease Genes and Manganese Poisoning*, SCI.DAILY (Feb. 2, 2009), *available at* http://www.sciencedaily.com/releases/2009/02/090201141559.htm (last accessed Aug. 17, 2010).

3A quick internet search of scientific studies published in 2007 shows that a considerable number of studies existed at the time, attempting to establish, with varying degrees of success, that manganese exposure among welders could cause Parkinson's Disease.

Case No. 1:04-CV-18948
UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF OHIO,
EASTERN DIVISION

JEFFREY & TERRY TAMRAZ, Plaintiffs,

v.

BOC GROUP, INC., ESAB GROUP, INC., HOBART BROTHERS CO., LINCOLN ELECTRIC CO., and TDY INDUSTRIES, INC., Defendants

July 18, 2008, Decided
July 18, 2008, Filed

KATHLEEN McDONALD O'MALLEY, UNITED STATES DISTRICT JUDGE.

MEMORANDUM & ORDER

This product liability action was brought by husband-and-wife plaintiffs Jeff and Terry Tamraz against the following five defendants, all of whom manufactured welding rods that Mr. Tamraz allegedly used: (1) BOC Group, Inc., (2) ESAB Group, Inc., (3) Hobart Brothers Company, (4) Lincoln Electric Company, and (5) TDY Industries, Inc. The case was assigned to the undersigned as related to the Multi-District Litigation known as *In re Welding Fumes Products Liability Litigation*, MDL No. 1501, case no. 03-CV-17000. Trial of this matter began on November 5, 2007 and culminated on December 5, 2007 with a jury verdict in favor of the plaintiffs. Specifically, the jury found: (1) against all five defendants on Mr. Tamraz's claims of

strict liability and negligent failure to warn, and awarded him $ 17.5 million in compensatory damages; (2) against all five defendants on Mrs. Tamraz's loss of consortium claim, and awarded her $ 3.0 million in compensatory damages; and (3) in favor of all five defendants on Mr. Tamraz's claims of fraudulent concealment and punitive damages. On December 6, 2007, the Court entered judgment consistent with this verdict. [1]

Twice during the course of trial - after the close of plaintiffs' case, and then after the close of their own case - defendants moved for judgment as a matter of law, pursuant to Fed. R. Civ. P. 50(a). The Court denied these motions. [2] Defendants now renew their earlier motions for judgment as a matter of law, pursuant to Fed. R. Civ. P. 50(b). The Court rules on these motions as follows:

> Defendants' Renewed Motion for Judgment as a Matter of Law on Causation (docket no. 174) is DENIED.
>
> Defendants' Renewed Motion for Judgment as a Matter of Law on Overexposure (docket no. 175) is DENIED.
>
> BOC's Motion for Judgment as a Matter of Law on Product Identification/Causation (docket no. 170) is GRANTED.
>
> ESAB's Renewed Motion for Judgment as a Matter of Law on Product Identification/Causation (docket no. 171) is DENIED.

Hobart's Renewed Motion for Judgment as a Matter of Law on Product Identification/Causation (docket nos. 172 & 176) is DENIED.

TDY's Renewed Motion for Judgment as a Matter of Law on Product Identification/Causation (docket no. 173) . is DENIED.

I. Legal Standards.

Federal Rule of Civil Procedure 50(a) states that, "[i]f a party has been fully heard on an issue during a jury trial and the court finds that a reasonable jury would not have a legally sufficient evidentiary basis to find for the party on that issue," then the court may "grant a motion for judgment as a matter of law against the party on a claim or defense that, under the controlling law, can be maintained or defeated only with a favorable finding on that issue." If, as did the undersigned in this case, the Court denies the Rule 50(a) motion made during trial, "the court is considered to have submitted the action to the jury subject to the court's later deciding the legal questions raised by the motion." [3] The movant may then "renew its request for judgment as a matter of law by filing a motion no later than 10 days after the entry of judgment." [4] The defendants timely filed their motions for judgment pursuant to Rule 50(b). [5]

After a verdict is returned, the Court may rule on a renewed Rule 50(b) motion by: (a) allowing the judgment to stand, (b) ordering a new trial, or (c) directing entry of judgment as a matter of law. [6] In

diversity cases, such as this one, the standard for granting a renewed motion for judgment as a matter of law under Rule 50(b) is governed by state law. [7] Accordingly, this Court must apply the standards of California law to defendants' motions.

Under California law, the "trial court's discretion in granting a motion for judgment notwithstanding the verdict is severely limited." [8] The trial judge cannot re-weigh the evidence, nor assess the credibility of witnesses. "If the evidence is conflicting or if several reasonable inferences may be drawn, the motion for judgment notwithstanding the verdict should be denied." [9] The motion "may properly be granted only if it appears from the evidence, viewed in the light most favorable to the party securing the verdict, that there is *no* substantial evidence to support the verdict. If there is *any* substantial evidence, or reasonable inferences to be drawn therefrom, in support of the verdict, the motion should be denied." [10] As another California appellate court has explained:

> We emphasize that the test is *not* the presence or absence of a substantial conflict *in the evidence*. Rather, it is simply whether there is substantial evidence *in favor of the* [*non-movant*]. If this "substantial" evidence is present, no matter how slight it may appear in comparison with the contradictory evidence, the judgment must be upheld. As a general rule, therefore, we will look only at the evidence and reasonable inferences supporting the successful party, and disregard the contrary showing. [11]

II. Analysis.

A. Causation.

With their first post-judgment motion, defendants argue the jury did not have a legally sufficient evidentiary basis to find in favor of the Tamrazes because the "plaintiffs did not adequately establish that defendants' failure to warn Mr. Tamraz about the risks of overexposure to welding fumes was the proximate cause of Mr. Tamraz's alleged injury." [12] In particular, defendants assert the Tamrazes "failed to prove at trial that Mr. Tamraz would have ceased welding or would have welded differently if a different warning had been included with the welding consumables he used." [13] The Court finds this assertion is not supported by the evidentiary record.

As an initial matter, the parties disagree on the question of the California legal standard for showing proximate cause in a failure-to-warn case. Defendants assert that, under California law, to prevail on a claim for failure to warn, a plaintiff "must prove not only that no warning was provided or the warning was inadequate, but also that the inadequacy or absence of the warning caused plaintiff's injury." [14] More specifically, defendants assert, the plaintiff bears the burden of showing at trial "that, had a different or better warning been given, plaintiff would have altered his or her behavior and avoided the injury alleged." [15] In further support of this position, defendants cite *Ramirez v. Plough, Inc.*; [16] Massok v. Keller Inds., Inc.; [17] and *Plummer v. Lederle Laboratories, Div. of American Cyanamid Co.* [18]

In contrast, the Tamrazes assert they bear no burden of adducing evidence that, if the defendants had supplied a

different or better warning, Mr. Tamraz would have behaved differently and taken action to avoid his alleged injuries. Essentially, the Tamrazes assert they should be given the benefit of the "heeding presumption," which is set out in the *Restatement Second of Torts*, §402A, comment j (1965): "Where a warning is given, the seller may reasonably assume that it will be read and heeded; and a product bearing such a warning, which is safe for use if it is followed, is not in defective condition, nor is it unreasonably dangerous." The heeding presumption "is a rebuttable presumption that is a burden-shifting device assisting a plaintiff in establishing causation." [19] As the court explained in the seminal case of *Reyes v. Wyeth Laboratories*: "Where a consumer, whose injury the manufacturer should have reasonably foreseen, is injured by a product sold without a required warning, a rebuttable presumption will arise that the consumer would have read any warning provided by the manufacturer, and acted so as to minimize the risks. In the absence of evidence rebutting the presumption, a jury finding that the defendant's product was the producing cause of the plaintiff's injury would be sufficient to hold him liable." [20]

As the Court noted when it addressed defendants' Rule 50(a) oral motion on causation, [21] it is not clear whether California law would recognize the heeding presumption in the context of this case. It does appear that "no California court has adopted or applied the heeding presumption." [22] However, the question of whether the presumption exists under California law has virtually always arisen in the context of medical device and drug cases. These cases implicate physicians, who are learned intermediaries, as the

intended warning recipients - the cases do not, like this one, involve consumer products with warnings directed at the end-user. [23] Further, in all of the cases cited by defendants (*Motus*, *Ramirez*, *Massok*, and *Plummer*), the plaintiff (and/or her doctor) did not read any warning at all, unlike Mr. Tamraz. This is a critical fact that makes these cases all distinguishable.

Ultimately, however, to rule on defendants' motion, the Court need not resolve the question of whether California law applies the heeding presumption in the context of this case. For the purpose of instructing the jury, the Court *rejected* the Tamrazes' argument that they were entitled to the heeding presumption. To the contrary, the Court accepted the defendants' position and instructed the jury as follows:

> The burden is on Mr. Tamraz, accordingly, to show, by a preponderance of the evidence, that the warnings given were inadequate, that an adequate warning would have made a difference in his conduct, and that this change in conduct would have prevented him from being injured. In other words, Mr. Tamraz must show that, if he had received different or additional warnings from the defendants, it is more likely than not that he would have altered his conduct in the use of welding consumables, and this alteration would have prevented him from suffering the same amount of harm. [24]

Even though this instruction did not give Mr. Tamraz the benefit of any heeding presumption, the jury found he would have heeded a better warning. Thus, defendants' present motion challenges whether Mr.

Tamraz presented sufficient evidence for a jury to reasonably find that Mr. Tamraz carried his burden of proof, even absent any presumption.

Having reviewed the evidence, the Court now concludes, as did the jury before it, that, even in the absence of any presumption, the Tamrazes produced sufficient evidence of proximate cause; they have made a showing adequate to support a finding by a reasonable jury "that Mr. Tamraz would have ceased welding or would have welded differently if a different warning had been included with the welding consumables he used." [25]

To make a sufficient evidentiary showing on this issue, there need not be uttered by plaintiff (or any other witness) certain "magic words;" counsel is not required to ask the plaintiff explicitly whether he would have behaved differently if he had received a different warning, and the plaintiff need not testify precisely to this effect. Indeed, a jury could reasonably perceive such a formulaic exchange as weightless.

Rather, circumstantial evidence (and inferences reasonably reached therefrom) can supply the "legally sufficient evidentiary basis" necessary to fend off a motion for judgment notwithstanding the verdict. In this case, plaintiffs adduced at trial the following evidence: (1) Mr. Tamraz read the defendants' warnings urging him to "use adequate ventilation," and he saw this phrase throughout his career; [26] (2) Mr. Tamraz did his best to follow this warning; [27] (3) Mr. Tamraz believed that obtaining adequate ventilation was necessary to avoid injury to lungs and eyes; he was never warned that inadequate ventilation could lead to

permanent brain damage, and the possibility never occurred to him; [28] (4) Mr. Tamraz was instructed that certain welding jobs called for use of a respirator, such as jobs involving lead paint or asbestos; [29] (5) whenever Mr. Tamraz understood that the nature of the job did require the use of a respirator, he used one; [30] and (6) one of Mr. Tamraz's welding foremen stated that Jeff was a conscientious worker who followed all the safety rules and procedures he was given, and he compared favorably in this regard to other welders. [31]

As noted earlier, "[i]f there is any substantial evidence, or reasonable inferences to be drawn therefrom, in support of the verdict," then a Rule 50(b) motion must be denied. [32] The evidence summarized above provides a sufficient basis upon which a reasonable jury could infer, find, and conclude that: (a) Mr. Tamraz was generally a careful welder; (b) Mr. Tamraz did his best to follow the warnings he was given, including directions on when and whether to use respirators; and (c) if warned that welding carried a risk of permanent brain damage, and that this risk could be mitigated by taking certain additional precautions (e.g., wearing a respirator), then Mr. Tamraz would have changed his behavior and welded differently.

In sum, the Court concludes that defendants' motion for judgment as a matter of law, based on an insufficient evidentiary showing that the proximate cause of Mr. Tamraz's alleged injury was the failure to warn him fully about the risks of overexposure to welding fumes, is not well-taken.

B. Overexposure.

With their second post-judgment motion, defendants argue the jury did not have a legally sufficient evidentiary basis to find in favor of the Tamrazes because plaintiffs offered no evidence that Mr. Tamraz "was exposed to sufficiently high doses of manganese in welding fumes to have caused his alleged injury." [33] Put differently, defendants assert that Mr. Tamraz's evidentiary burden is not just a showing of exposure to manganese in welding fumes; he must show he was exposed to *toxic levels* of manganese. Further, defendants assert, there must be reliable expert testimony to support this showing. Defendants insist the Tamrazes did not meet this burden at trial.

California courts have set out the analysis this MDL court must undertake, in the toxic-tort product liability context, to determine whether there is sufficient evidence of exposure to a toxin to support a verdict. In *Lineaweaver v. Plant Insulation Co.*, the court relied upon "established California law and long-standing tort principles to resolve the single issue put to [it]: was there evidence of sufficient substantiality to support a jury finding that asbestos supplied by [the defendant] was a cause of [the plaintiffs'] injuries?" [34] The court explained it was "concerned with the element of causation and, in particular, the cause in fact component of that element." [35] The court continued:

> The "substantial factor" standard is used for cause in fact determinations. Under that standard, a cause in fact is something that is a substantial factor in bringing about the injury. * * *
>
> "Substantial factor" has not been judicially

> defined, and some think it "neither possible nor desirable to reduce it to any lower terms." However, it has been suggested that a force which plays only an "infinitesimal" or "theoretical" part is not a substantial factor. But the word "substantial" should not be weighed too heavily. The substantial factor standard [was] formulated to aid plaintiffs as a broader rule of causality than the "but for" standard . . . .[36]

The *Lineaweaver* court then held that, "in evaluating whether [the alleged] exposure was a substantial factor in causing [the claimed] disease, the standard should be the same as used in other negligence cases: is there a reasonable medical probability based upon competent expert testimony that the defendants' conduct contributed to plaintiff's injury[?]" [37] Other California courts have confirmed that, where the issue is "causation in a toxic tort case, expert testimony is required to prove, with 'reasonable medical probability,' that exposure to the defendant's products or emissions caused the plaintiff's injuries." [38] And, of course, "[f]actual assumptions made by an expert in arriving at an opinion [regarding toxic exposure levels] must have sufficient evidentiary support." [39] If an expert's "opinion is not based upon facts otherwise proved . . . it cannot rise to the dignity of substantial evidence." [40]

Finally, the factors a court should examine when assessing the medical probability that exposure to a toxin contributed to the plaintiff's disease include: "[f]requency of exposure, regularity of exposure, and proximity of the . . . product to [the] plaintiff," as well as "the type of . . . product to which plaintiff was exposed,

the type of injury suffered by plaintiff, and other possible sources of plaintiff's injury." [41]

In this case, the jury was presented with the following evidence, much of it adduced from plaintiffs' expert Industrial Hygienist, David Kahane. [42] Several governmental and professional entities have established various measures to define safe exposure limits to toxins, including manganese. These measures include: (1) Threshold Limit Values ("TLVs"), which are promulgated by the American Conference of Governmental Industrial Hygienists ("ACGIH"); (2) Permissible Exposure Limits ("PELs"), which are promulgated by the Occupational Safety and Health Agency ("OSHA"); and (3) additional PELs promulgated by the California state equivalent of OSHA, known as "Cal-OSHA." The TLV is the maximum *average* amount to which, it is believed, a person may be safely exposed over an 8-hour time period. [43] The PEL is the maximum *ceiling* amount to which a person may be safely exposed at any moment in time. Thus, a welder may suffer momentary manganese fume exposures in excess of the PEL ceiling limit, but not suffer an average exposure over his workday in excess of the TLV; similarly, a welder may suffer average exposures in excess of the 8-hour limit imposed by the TLV, but not momentary exposures in excess of the PEL absolute ceiling.

Over time, the ACGIH has reduced the TLV for manganese exposure. In 1948, the TLV was an 8-hour time-weighted average of 6.0 mg/mm<3>; in the late 1970s, when Mr. Tamraz started welding, the TLV was 5.0 mg/mm<3>; and today, it is an 8-hour time-weighted average of 0.2 mg/mm<3>. This decrease reflects the

ACGIH's increasing concern over the toxicity of manganese exposure. OSHA's current PEL for manganese, which has been in effect since the early 1970s, is 5.0 mg/mm<3>, while Cal-OSHA's current PEL for manganese exposure is 0.2 mg/mm<3>. OSHA's higher PEL incorporates concerns regarding the cost to businesses of compliance with exposure limits, while the ACGIH's TLVs are concerned exclusively with worker safety. [44]

Documents authored by the defendants, and by trade organizations to which the defendants belong, discuss manganese in welding fumes and the applicable PELs and TLVs - especially during times when the TLVs were lowered. One such document, circulated widely within the welding industry at about the time Mr. Tamraz started welding, is known as the Battelle Survey; it was sponsored by the American Welding Society in 1972. The Survey stated:

> The fumes from manganese are highly toxic, and they can produce total disablement *even after exposures as short as a few months* to high-fume concentrations . . . . Exposure to manganese dioxide may cause a neurological lesion involving the basal ganglia, the frontal cortex, and occasionally the pyramidal system. Symptoms are similar to Parkinson's syndrome and include 'weakness of the legs,' difficulty in walking downhill, instability, and weakness while doing heavy work. [45]

The Survey also stated that several welding rods "produced manganese and vanadium fumes that

exceeded recommended TLVs," which were 5.0 mg/mm<3> at the time. [46]

Documents authored by defendant Lincoln Electric concede that "early limits are outdated and insufficiently protective of worker health." [47] More specifically, minutes of a 1981 meeting of the American Welding Society ("AWS"), attended by Lincoln, state: "some cases of manganism have been documented at levels below five milligrams per cubic meter, and that this was the reason that the current threshold limit value was lowered to one milligram per cubic meter." [48] Further, defense witnesses admitted that the level at which manganese exposure becomes unsafe is not known for certain; [49] and plaintiffs' expert Kahane testified there is at least one "report of a documented Manganese-Induced Parkinsonism case that was purported to occur with levels below [the current TLV of] point two." [50]

In 1994, members of the AWS met to discuss the then-proposed reduction of the manganese exposure TLV from 1.0 to 0.2 mg/mm<3>. The AWS meeting minutes state that defendant Lincoln Electric "estimated that if the TLV is lowered to 0.2 milligrams per cubic meter, then the overall welding fume limit . . . would be exceeded in most workplace atmospheres." [51] As noted above, the reduction of the TLV to 0.2 mg/mm<3> did, in fact, occur. [52]

In addition to setting manganese exposure limits, OSHA agents travel to various worksites around the country to audit whether employees, including welders, are experiencing exposures in excess of the legal limits. These measurements are collected in an OSHA

database. OSHA's measurements of manganese exposures endured by welders includes all types of circumstances: indoor and outdoor welding, use of high- and low-manganese welding rods, well-and poorly-ventilated conditions, and so on. Analysis of the OSHA database - which includes thousands of measurements taken over the last 20 years - shows that: (1) about 30% of the welders were, at the time of measurement, experiencing manganese exposure in excess of OSHA's current TLV of 0.2 mg/mm<3>; (2) about 5% of the welders were, at the time of measurement, experiencing manganese exposure in excess of the ACGIH's current "excursion limit" (recommended absolute ceiling) of 1.0 mg/mm<3>; (3) welders were about five times as likely to be experiencing manganese exposure in excess of OSHA's current TLV as were non-welders; and (4) these statistics have held fairly constant over the duration of the 20-year measurement period. [53]

Beyond these generalized observations regarding welding fume exposures experienced by the "typical" or "average" welder, Mr. Tamraz and one of his foremen-supervisors, Mr. Montoya, testified regarding Mr. Tamraz's own welding experiences. This evidence may be summarized as follows: (1) because Mr. Tamraz was a very industrious welder with excellent skills, employers regularly requested his services; (2) during the majority of his career, Mr. Tamraz used "flux-cored wire," which is known as one of the "smokiest" welding consumable products and which produces fume with one of the highest manganese concentrations; (3) the amount of time that Mr. Tamraz spent welding during a workday (as opposed to related chores where he was not exposed to fumes) was often high; (4) at least

occasionally, Mr. Tamraz welded in tight confines where there was little natural ventilation available; and (5) Mr. Tamraz was only infrequently instructed to use a respirator - usually when the job involved welding near asbestos or on metals coated with lead-based paint. [54]

Finally, there was substantial evidence that: (1) excessive exposure to manganese can cause a disease known as Manganism, or Manganese-Induced Parkinsonism ("MIP"); (2) the symptoms of MIP are similar to those of Idiopathic Parkinson's Disease (which is what defendants' experts maintain Mr. Tamraz has); and (3) individual susceptibility to the adverse effects of manganese varies considerably. Indeed, defendants' own experts and documents concede these points. For example, the 1985 Material Safety Data Sheet that accompanied defendant Hobart's welding rods stated: "Long term overexposure to manganese compounds may affect the central nervous system. Symptoms include muscular weakness, tremors similar to Parkinson's disease." [55] ESAB's warning labels now state: "Overexposure to manganese, and manganese compounds above safe exposure limits can cause irreversible damage to the central nervous system, including the brain." [56] Dr. Anthony Lang, a defense expert neurologist specializing in movement disorders, agrees that welders can get Manganese-Induced Parkinsonism from welding fume exposure. [57] And a document published by the World Health Organization addressing environmental health criteria for manganese states that "[i]ndividual susceptibility to the adverse effects of manganese varies considerably." [58]

Despite this evidence, defendants argue that Mr. Tamraz did not present the jury with substantial evidence that his disease was caused by exposure to the fumes given off by defendants' welding consumables. Defendants assert Mr. Tamraz is required to show he was "exposed to a sufficient amount of the substance in question to elicit the health effect in question," which "requires not simply proof of exposure to the substance, but proof of *enough* exposure to cause the plaintiff's specific illness;" and that Mr. Tamraz did not meet this requirement. [59] The Court disagrees.

The essential prongs of defendants' argument are: (1) there is some low level of exposure to manganese that is certainly safe; (2) it is not known precisely at what level manganese exposure causes Manganese-Induced Parkinsonism; (3) the plaintiffs did not show precisely how much manganese fume exposure Mr. Tamraz actually sustained; (4) the only objective, quantitative evidence of fume exposure presented by plaintiffs was the OSHA database, and there is no basis upon which to conclude the welders with scores at the "high end" of this database had work conditions and exposures similar to Mr. Tamraz; (5) Mr. Tamraz's anecdotal, subjective descriptions of his welding fume exposures is self-serving, and does not allow for any quantification; and (6) therefore, a conclusion by the jury that Mr. Tamraz was exposed to toxic levels of manganese fume must be speculation.

This argument is flawed because defendants have refused to recognize the inferences a reasonable jury may (and, apparently, did) draw from the evidence presented. It is true that Mr. Tamraz did not present at trial any air sampling data that showed, at any point

during his career, the actual concentrations of manganese fume in his breathing space. Of course, virtually no welder could ever provide this evidence, which is why the law does not require mathematical precision to show toxic exposure. As stated by the *Reference Manual on Scientific Evidence*, "[o]nly rarely are humans exposed to chemicals in a manner that permits a quantitative determination of adverse outcomes . . . . Human exposure occurs most frequently in occupational settings where workers are exposed to industrial chemicals like lead or asbestos; however, even under these circumstances, it is usually difficult, if not impossible, to quantify the amount of exposure." [60] Rather, Mr. Tamraz was required only to show "with 'reasonable medical probability,' that exposure to the defendant's products or emissions caused [his] injuries." [61]

The case of *Bonner v. ISP Technologies, Inc.* is instructive. [62] In *Bonner*, the Eighth Circuit Court of Appeals analyzed the quantum of proof of exposure necessary to show causation in a toxic tort case. After plaintiff Bonner was splashed with an organic solvent known as "FoamFlush," she suffered cognitive impairment and Parkinsonian symptoms. The jury awarded her $ 2.2 million in damages, and the defendant manufacturer appealed, asserting (among other things) that there was insufficient evidence of exposure at toxic levels to support the verdict. The Circuit Court affirmed the judgment, stating that "Bonner did not need to produce a mathematically precise table equating levels of exposure with levels of harm in order to show that she was exposed to a toxic level of FoamFlush, but only evidence from which a reasonable person could conclude that her exposure

probably caused her injuries." [63] The court further held that the evidence was sufficient to support the jury's verdict even though Bonner's expert "was unable to offer a threshold exposure amount for injury to occur." [64] The court explained:

> it was not necessary that Bonner's experts quantify the amount of FoamFlush to which she was exposed in order to demonstrate that she was exposed to a toxic level of BLO. It is sufficient for a plaintiff to prove that she was exposed to a quantity of the toxin that "exceeded safe levels." Bonner presented witnesses who testified that [1] her exposure to FoamFlush was of a duration and of a volume sufficient to support a conclusion that she inhaled and/or absorbed through her skin at least a quarter of a teaspoon of FoamFlush when she was sprayed with it [and [2]] ingested doses of GHB, BLO's metabolite, as small as a quarter of a teaspoon can have toxic effects, and that inhalation is a more potent exposure mechanism than is ingestion. [65]

The same analysis applies in this case. Mr. Tamraz was not required to quantify the amount of manganese fume he was exposed to; he was required only to prove he was exposed to amounts that exceeded safe levels. This proof, like Bonner's, included testimony from the Mr. Tamraz, himself, and also his co-worker, regarding his "[f]requency of exposure, regularity of exposure, and proximity of the . . . product to [the] plaintiff," as well as "the type of . . . product to which plaintiff was exposed, the type of injury suffered by plaintiff, and other possible sources of plaintiff's injury." [66] Specifically, Mr.

Tamraz offered proof at trial that the products he used emitted fumes with high manganese levels; the frequency and regularity of his exposure to manganese fumes were both high; [67] he was occasionally in situations with little ventilation; and his symptoms are entirely consistent with excessive manganese exposure. Even though none of the parties' experts could define the threshold exposure amount for injury to occur, the only expert industrial hygienist who offered any opinion at trial [68] opined that Mr. Tamraz was regularly and certainly exposed to amounts that *exceeded safe levels*, as defined by the current TLV. And Mr. Tamraz's expert neurologist agreed with his treating neurologist, opining that these excessive exposures caused his symptoms. These opinions enjoyed substantial evidence to support them, and that is all this Court must find to sustain the verdict.

In sum, the Court easily concludes that there was presented at trial substantial evidence to support the opinions of Mr. Tamraz's experts that his exposure to the fumes given off by defendants' products was sufficient to, and did, cause his injuries, with reasonable medical probability. Indeed, given that defendants' own documents discuss the substantial likelihood that welders in "most workplace atmospheres" will experience manganese exposure in excess of the threshold safety level of 0.2 mg/mm<3> TLV, and given that about one third of the welders in the OSHA database were, at the time of measurement, experiencing manganese exposure in excess of the same TLV, any inferences and assumptions made by Mr. Tamraz's experts (and by the jury), based on the evidence at trial of Mr. Tamraz's actual workplace

conditions and exposure, also had substantial evidentiary support. [69]

For these reasons, defendants' motion for judgment as a matter of law, based on insufficient evidence of overexposure to welding fumes, is not well-taken.

C. Product Identification.

Both Mr. and Mrs. Tamraz obtained a verdict against five defendants: (1) BOC Group, Inc., (2) ESAB Group, Inc., (3) Hobart Brothers Company, (4) Lincoln Electric Company, and (5) TDY Industries, Inc. For each plaintiff, however, the jury entered only a single award of compensatory damages, payable jointly from all five defendants - as opposed to five separate awards, payable by each defendant individually. This result was requested and agreed to by the defendants before the Court charged the jury. [70]

With their third set of post-judgment motions, four of the five defendants - all but Lincoln Electric - argue there was insufficient product identification by Mr. Tamraz to support the jury's verdict against them. For example, defendant BOC argues that "plaintiffs failed to carry their burden of establishing that Mr. Tamraz was exposed to a product *manufactured by BOC*," as opposed to a welding consumable manufactured by some other entity. [71] Some of the defendants add that there was insufficient identification by Mr. Tamraz of which of their *particular* warnings were inadequate, so that the jury's verdict against them cannot stand. If the Court granted all of the post-judgment motions filed by the four defendants on these bases, Lincoln Electric would be left alone to satisfy the judgment.

To give context to the defendants' motions, the Court first reviews the nature of the welding products used by Mr. Tamraz and the warnings that accompanied them.

1. Background.

The process of welding generally involves the melting of a "welding consumable," which is used to join two pieces of metal. Welding consumables come in two categories: (1) stick electrodes, which are relatively short; and (2) wire, which comes on spools in longer lengths. When using stick electrodes, the welder has to pause periodically to attach a new stick to his welding apparatus; when using wire, the welder pauses to "reload" much less frequently.

The American Welding Society has created a standardized numbering identification system applicable to all welding consumables. For example, a welding stick identified as an "E7018" is: (a) an electrode ("E"); (b) that will produce a weld with a tensile strength of 70,000 p.s.i. ("70xx"); (c) which may be used in all welding positions, including overhead ("xx1x"); and (d) can be used with both direct and alternating current welding machines ("xx18"). As to wire consumables, welders often refer to wire products by the welding process for which they are used. For example, welders may refer to a wire consumable as "MIG" - an acronym for Metal Inert Gas, which is a welding process involving automatic feeding of a continuous, wire consumable shielded by an externally-supplied gas. To a large extent, welding consumables, both electrodes and wire, are fungible - that is, many manufacturers produce E7018 sticks, or MIG wire.

When asked to identify what welding products he used, Mr. Tamraz responded with both standardized AWS identification numbers and also trade names. For example, as discussed further below, Mr. Tamraz testified he used "7018, 6010, [and] 6011" electrodes. He also stated he used "Innershield wire," referring to a flux-cored wire product (AWS identification number E71T-11) manufactured under the "Innershield" trade name by Lincoln Electric.

With regard to the warnings employed by the defendants during the course of Mr. Tamraz's career, the evidence showed the warning language did not vary from one defendant to another, even as it evolved. In some cases, there were very minor wording differences from one manufacturer's label to another; for the most part, however, all of the manufacturers' warnings adhered to the AWS standards, and changed 25 in lockstep. [72]

Specifically, before 1967, none of the defendants supplied any warning with their welding consumables. In 1967, the AWS adopted a mandatory standard warning label, which all five defendants promptly started to use. The 1967 AWS warning stated:

> Caution. Welding may produce fumes and gases hazardous to health. Avoid breathing these fumes and gases. Use adequate ventilation. See USAS Z49.1, 'Safety in Welding & Cutting' published by the American Welding Society. [73]

In addition to AWS documents discussing the adoption of this standard warning, the jury was shown evidence of this language appearing on the labels that

accompanied Hobart, BOC, and Lincoln products. [74] Counsel for defendants referred repeatedly during trial to "the 1967 warning" that was on the welding consumables used by Mr. Tamraz early in his career, with the understanding that all defendants were supplying the same warning. [75] Although this warning urged welders to avoid breathing the fumes and gases produced during welding, it did not warn that overexposure to manganese in the fumes could cause permanent brain damage.

In 1979, the AWS adopted a new, mandatory standard warning label. Again, all five defendants changed their label to match the AWS's mandates, although not until about 1981. The 1979 AWS warning stated, in pertinent part:

> FUMES AND GASES can be dangerous to your health.
>
> - Keep your head out of fumes.
> - Use enough ventilation or exhaust at the arc or both.
> - Keep fumes and gases from your breathing zone and general area.
>
> * * *
>
> See American National Standard Z49.1, "Safety in Welding and Cutting," published by the American Welding Society.

As with the 1967 warning, counsel for defendants made clear that the 1979 warning was used by all defendants. [76] In addition to AWS documents discussing the adoption of this standard warning, the jury was shown evidence of this language appearing on the labels that accompanied Lincoln products. [77] As before, this warning directed welders to avoid breathing welding fumes, but did not warn that failure to do so could cause permanent brain damage.

Neither plaintiffs nor defendants adduced any evidence regarding the warnings used by the defendants after the "1979 warning." Thus, although it was suggested to the jury that some of the defendants modified their welding consumables warnings again in 1985, the jury was not presented with evidence of this subsequent change in language. [78]

At trial, Mr. Tamraz argued that the defendants' 1967 and 1979 welding consumable warnings were inadequate in several respects. For example, Mr. Tamraz and his experts criticized certain defendants for placing warnings where they were not likely to be seen; disparaged certain labels because they were too small to read; and faulted all labels for referring to other documents, such as the lengthy ANSI Standard Z49.1, rather than setting out warning language in the label, itself. But Mr. Tamraz's principal, overriding criticism of *every* warning issued by *every* defendant - regardless of when issued - was directed at the label's content, or lack thereof. Specifically, Mr. Tamraz's chief argument at trial was that *all* of the labels that accompanied *all* of the products he used during *all* of his career were inadequate, because the labels did not warn explicitly that exposure to welding fumes could cause permanent,

irreversible brain damage that becomes progressively worse over time. And the evidence adduced at trial was that, in fact, none of the defendants' labels available to Mr. Tamraz during his career did contain any such explicit warning. [79]

2. Applicable Law.

As this MDL Court has recognized, "[i]t is elementary that in any action claiming injury from a product, the plaintiff must show causal connection between the defendant manufacturer and that product." [80] California law is in accord: "Proximate cause is a necessary element of actions for both negligence and strict liability." [81] In the context of product liability claims, this means that, "to hold a producer, manufacturer, or seller liable for injury caused by a particular product, there must first be proof that the defendant produced, manufactured, sold, or was in some way responsible for the product." [82] If a plaintiff cannot adduce sufficiently particularized evidence of exposure to the defendant's product, then "there is no causation." [83]

A review of several California cases cited by defendants reveals how these rules are applied in practice. In *McGonnell v. Kaiser Gypsum Co.*, [84] plaintiff McGonnell was a plumber/pipefitter who worked at a Medical Center for 24 years; his duties included cutting through walls and pipes, where he would encounter insulation and fireproofing materials. After McGonnell contracted asbestosis, he sued Kaiser Gypsum and Kaiser Cement, asserting he was exposed to asbestos-containing products they manufactured that were present at the Medical Center where he worked. McGonnell testified that: (1) he "had no knowledge of exposure to products

manufactured by [either defendant]; (2) "as far as he knew, he had never worked with Kaiser Gypsum's products or near others using Kaiser Gypsum products"; and (3) "he had heard of Kaiser Cement Company and seen bags of cement with that name on it, but he could not recall where he had seen the bags." [85] Despite this testimony, McGonnell pressed his claims, offering evidence (such as invoices) that "showed products manufactured by Kaiser Gypsum might have been delivered to [the] Medical Center . . . and possibly used in building additions or renovations" and showed further that "plastic cement [manufactured by Kaiser Cement] might have been used in construction at [the Medical Center]." [86]

In assessing this evidence of exposure to the defendants' products, the *McGonnell* court observed that "McGonnell was one of the best persons, if not the best person, to identify the various products and substances to which he had been exposed during his employment." [87] Having failed to "place any Kaiser products at his place of employment" through direct recollection, it became McGonnell's burden to "produce some circumstantial evidence to establish exposure to Kaiser products." [88] The court, viewing all of the evidence in a light most favorable to McGonnell, agreed that "it is at least within the realm of possibility that McGonnell encountered a wall with Kaiser [Gypsum] joint compound during his 24 years of employment at [the Medical Center]," and that "Kaiser Cement products might have been used once on a construction project [there]." [89] But this evidence was not substantial enough to avoid judgment as a matter of law. The court granted summary judgment to the defendants, concluding: "it is not enough to produce just *some*

evidence [of exposure to defendants' products]. The evidence must be of sufficient quality to allow the trier of fact to find the underlying fact in favor of [the plaintiff]. All that exists in this case is speculation that at some time McGonnell might have cut into a wall that might have contained Kaiser joint compound that might have contained asbestos." [90] The *McGonnell* court did not set the evidentiary bar high, but concluded that the circumstantial evidence and inferences presented by McGonnell failed to clear the modest legal hurdle.

Defendants also cite *Dumin v. Owens-Corning Fiberglas Corp.*, [91] which contains a similar analysis. In *Dumin*, the plaintiff could not "identify [defendant Owen-Corning's product] Kaylo as one of the asbestos products to which he was exposed, but instead relie[d] upon circumstantial evidence to establish exposure." [92] Viewed "in its best light," the plaintiff's circumstantial evidence was that: "(1) [he] was aboard the [ship] Pocono in 1953 and 1954, and his duties included making any necessary boiler repairs with insulation materials; (2) the Pocono was home ported at the Norfolk Naval Shipyard; (3) Kaylo was used at the Norfolk Naval Shipyard as one of many asbestos insulation products at some time around the early 1950's; and (4) the Pocono's repair supplies were probably received from the Norfolk Naval Shipyard." [93] The question presented to the appellate court was "whether this evidence would support a jury determination that [the plaintiff] was exposed to [Owens-Corning's] Kaylo, since [he] must establish exposure to prove that his asbestos disease was caused by [Owens-Corning's] conduct in distributing its product." [94] The court affirmed the trial court's entry of a directed verdict because "a conclusion that [the plaintiff] was exposed to . . . Kaylo while

aboard the Pocono in 1953 and 1954 would require a stream of conjecture and surmise." [95] Without a stronger inferential basis upon which to conclude that the plaintiff suffered actual exposure to the defendant's product, the law required a verdict be directed in Owens-Corning's favor.

Defendants next cite *Garcia v. Joseph Vince Co.*, [96] a case with facts worthy of a law school exam. In *Garcia*, the plaintiff's "eye was injured when an opponent's sabre broke through a fencing mask worn by [plaintiff] during a fencing bout." [97] After the accident, the sabre was examined and then "placed back in the team bag" containing other blades; "the identity of the particular blade in question was thereby lost." [98] All parties agreed that the sabre had been manufactured by one of two defendant companies, known as "American" and "Vince," but "which one of the two was unknown. There was no evidence that the blade was in fact known to have been manufactured by American or to have been manufactured by Vince." [99] At the close of evidence, the trial court entered judgment in favor of the defendants. The appellate court affirmed, noting that, to hold a defendant "liable for injury caused by a particular product, there must first be proof that the defendant produced, manufactured, sold, or was in some way responsible for the product." [100] The *Garcia* court concluded that, in the peculiar circumstances presented, "[t]he jury on the basis of [the] evidence would be purely speculating as to who should be liable." [101] Without some valid evidentiary basis for finding *which* defendant had actually manufactured the blade that caused the plaintiff's injury, both defendants were entitled to a directed verdict. [102]

A final instructive case is *Lineaweaver v. Plant Insulation Co.*, [103] in which three different plaintiffs -- Ward, King, and Lineaweaver -- sought damages due to asbestos exposure. The trial court granted a directed verdict in favor of defendant Plant, "which was the exclusive Northern California distributor of Fibreboard insulation products, marketed under the Pabco trademark," and against all three plaintiffs. [104] After examining the evidence each plaintiff supplied of his exposure to Pabco, the appellate court affirmed as to plaintiffs Ward and King, but reversed as to Lineaweaver.

In reviewing the evidence, the *Lineaweaver* court found that none of the plaintiffs had offered testimony that he used or was exposed to Pabco while on the job; they all presented only circumstantial evidence that Pabco was present at their worksites and they were exposed to it. The appellate court went on to take careful measure of the substantiality of this circumstantial evidence. As to plaintiff Ward, the evidence "only show[ed] that Pabco may have been minimally used as a 'fill-in' at uncertain times aboard one out of every three or four of the approximately 100 ships serviced" at the shipyards where he worked. [105] This did not amount to substantial evidence that "Pabco asbestos products were actually used, or even probably used, on any of the ships at the time Ward was serving aboard them." [106] Plaintiff King offered similar evidence, which the court found insubstantial: "Even accepting this evidence as sufficient to permit an inference that some amount of Pabco products were [*present*] at [the shipyards where King worked] during the time of King's employ, the evidence is wholly inadequate to support the conclusion that King was *exposed* to Pabco.

It would be unreasonable to infer that King was exposed to Pabco asbestos products which were only incidentally used at the sprawling shipyards." [107]

The court's calculus, however, was different with respect to plaintiff Lineaweaver, because his circumstantial evidence of actual exposure was stronger. In reaching this conclusion, the *Lineaweaver* court was scrupulous in allowing for reasonable inferences in Lineaweaver's favor, and careful to distinguish *Dumin*:

> Lineaweaver . . . presented sufficient evidence of exposure to Plant[-]supplied asbestos products, as follows: (1) Plant was the exclusive distributor in Northern California of Pabco asbestos insulation products beginning in 1948; (2) Lineaweaver worked at the Standard Oil refinery from 1950 to 1984, repeatedly working with and around asbestos insulation; (3) Lineaweaver worked throughout the sprawling refinery which has insulation over about two-thirds of its pipes and much of its equipment; (4) Lineaweaver saw boxes of Pabco products at the refinery; (5) Plant was a significant supplier of asbestos products, performing about 50 percent of the insulating work at the refinery in the 1960's; [and] (6) another major insulation contractor used Pabco and another product as "fill-in" supplies which constituted 10 to 15 percent of the refinery's insulation installed by that contractor. While there was no direct evidence that Lineaweaver was exposed to Plant[-]supplied Pabco, the circumstantial evidence was sufficient to support a reasonable

> inference of exposure. Unlike [*Dumin*], in which we found insufficient evidence of exposure to a particular asbestos product, plaintiff has established that defendant's product was definitely at his work site and that it was sufficiently prevalent to warrant an inference that plaintiff was exposed to it during his more than 30 years of working with and around asbestos throughout the refinery. [108]

The *McGonnell*, *Dumin*, *Garcia*, and *Lineaweaver* cases [109] all make clear that, "[u]ltimately, the sufficiency of the evidence of causation will depend on the unique circumstances of each case." [110] If a plaintiff offers only weak, circumstantial evidence of exposure to a defendants' product, "creat[ing] a dwindling steam of probabilities that narrow into conjecture," then judgment as a matter of law is appropriate. [111] If, however, the plaintiff offers at trial "evidence sufficient to support a reasonable inference of exposure" to the defendant's product, then a motion for judgment as a matter of law must be denied. [112]

3. Tamraz's Evidence of Exposure to Defendants' Products.

Unlike the cases cited in the preceding discussion, the tradesman-plaintiff in this case offered direct evidence of his exposure to the defendants' products. Specifically, plaintiffs' counsel asked Mr. Tamraz at trial to identify the companies who manufactured the welding consumables he used during his career. The initial colloquy on this subject is set out below.

> Q. Were you welding in 1992?

A. Yes.

Q. At times, did you use welding consumables manufactured by ESAB?

A. Yes.

Q. What other manufacturers' consumables did you use?

A. Well, that I can recall, I used Lincoln quite a bit. I used Hobart, Airco, Linde, ESAB, and McKay. That's about all I can remember.

Q. Thank you. [113]

This colloquy, by itself, distinguishes this case from those cited by defendants and discussed above. In none of those cases did the plaintiff, himself, provide direct testimonial evidence that he used or was exposed to the products in question. Here, Mr. Tamraz stated explicitly that he used welding consumables manufactured by defendants ESAB, Hobart, and Lincoln. He also stated: (1) he used "McKay" welding consumables, and it is not disputed that defendant TDY is responsible for products distributed under this brand name before 1992; [114] and (2) he used Airco welding consumables, and it is not disputed that defendant BOC is responsible for products distributed under this brand name through 1986. [115]

While it might fairly be argued that this colloquy, alone, provides substantial evidence that Mr. Tamraz was exposed to manganese fumes emitted by products manufactured by at least some of the five defendants,

plaintiff's counsel adduced at trial additional testimony from Mr. Tamraz on this subject, set out here at length:

> Q. Just so we're clear, I asked you this early on, but I would like to take the liberty and ask you again. Tell the jury the different welding manufacturers whose products you used. I believe you said Lincoln, Airco, Teledyne, ESAB?
>
> A. Lincoln, Airco, McKay, ESAB, Hobart, Linde. I have used more, but that's all I can remember.
>
> Q. Are you familiar with something called CE Toland?
>
> A. Yes.
>
> Q. What is that?
>
> A. It is a company I worked for.
>
> Q. And on that job, do you remember particular consumables that you were using?
>
> A. Oh, yeah.
>
> Q. Which ones?
>
> A. We used Lincoln 7018 along with, I think, Hobart 7018. That is this rod here. It's a good electrode. It's an excellent rod. I used that on the Toland job, and I used Lincoln Innershield, NR211. And then R232, that's the spool here.

Q. Did you work for a company called Cabrillo Hoist?

A. Yes, I did.

Q. Where did you work for them?

A. Oh, throughout my whole career. Every time I worked for them it was basically just stick welding.

Q. Do you remember --

A. They erected man lifts, the exterior elevators. They are temporary elevators you see, they are orange. They call them man lifts. They take the construction workers up and down. As the job progresses, eventually they take the elevator down. Man erection they call it.

Q. Do you remember the names of the products?

A. With them I used fleetweld 5, 6011.

Q. Who manufactured that?

A. Lincoln.
Q. Any other?

A. And I used Lincoln 7018.

Q. Anything else? Any other defendant products, ESAB, Airco, Teledyne?

A. I can't recall that, for that particular company.

Q. How about Lee's Imperial Welding?

A. Yes.

Q. What jobs did you work for them, if you recall?

A. I worked on the Great Mall, the big Ford Motor Company plant, the Ford Motor Company. It was an enormous plant they shut down. Actually, we went in there and retrofitted the job. We used a lot of NR211 Lincoln.

Q. Mejia Steel?

A. Mejia.

Q. Whose product do you recall?

A. We used Lincoln NR232 the whole job, and also Lincoln 7018. That was all they would let us touch in that freeway overpass.

Q. How about the ENDI Metal Works?
A. I worked for them. That was a good company. I worked for them on and off for years. What do you want to know?

Q. Same thing. Which products?

A. For ENDI, I used P-5 or 5P6010.

Q. Give us the name of the manufacturers, do you recall?

A. The name of the manufacturer?

Q. Yes.

A. Lincoln, Hobart, Airco.

Q. Any others?

A. Linde, McKay. This is back earlier in my career, yes.

Q. Schrader Iron Works, are you familiar?

A. Yes.

Q. Do you remember the product?

A. Lincoln NR211, a lot of 7018, Lincoln. A lot of P56010 and N6011 stick. [116]

Plaintiffs' counsel then concluded his direct examination of Mr. Tamraz by showing a picture of the logos of all five of the defendants' welding products:

Q. Look up at the screen. I think you have testified that you used the products of every one of those defendants whose logos are on the screen, correct?

A. Yes, that's right. Teledyne McKay, is that the same one? The same product, right? Is that the same name?

Q. Teledyne McKay?

A. Yes. I have used every one. [117]

In addition to the product identifications adduced during his direct testimony, Mr. Tamraz also testified as follows on cross-examination by defense counsel:

Q. Could you identify for me what products of these defendants you utilized?

A. What products?

Q. What products.

A. You mean like this?

Q. Yes. What products?

A. You mean the names of the companies?

Q. Companies and identify the products.

A. You have the Lincoln Innershield wire, Lincoln on stick wire, Lincoln stick electrode. You have Hobart stick; I think I have used some Hobart MIG.

Q. Can you be more specific with the actual product type?

A. What are you getting at? I don't understand.

Q. You know, the number on the product.

A. The number of the product? You mean like the 7018s?

Q. Yes.

A. I have used Lincoln NR311, NS3M, .120 Innershield wire, 7018, 6010, 6011, stuff like that.

Q. Anything else from other manufacturers?

A. Yeah. I think I used some McKay 30816 lime coat for stainless steel welding.

Q. Any other companies?

A. Airco 7018s, ESAB - I think the ESAB was stick. I'm not sure which one it was.

Q. Any other companies?

A. I have used some, I think Linde 7014 stick.

Q. No trouble remembering the products you used from these companies?

A. Well, yeah. There is trouble. I mean, I can't remember them to the T, but yeah.

Q. Okay. But you can remember –

A. I'm pretty sure. [118]

Finally, in addition to Mr. Tamraz's testimony, a representative from ESAB arguably *conceded* that Mr. Tamraz used their welding products. [119]

The question presented by the defendants' motions is whether this testimony is "substantial evidence" that Mr. Tamraz was exposed to their products. The Court answers this question for each defendant, below. [120]

4. Analysis.

a. ESAB.

As revealed by the testimony quoted above, Mr. Tamraz stated to the jury four different times that he used welding consumables manufactured by ESAB. One of these identifications was slightly more specific -- "I think the ESAB was stick. I'm not sure which one it was." In contrast to Mr. Tamraz's more-specific testimony about Lincoln products, however -- for example, "I have used Lincoln NR311, NS3M, .120 Innershield wire, 7018, 6010, 6011, stuff like that" -- Mr. Tamraz did not identify exactly which ESAB welding consumables he used. ESAB asserts Mr. Tamraz's testimony thus remains insufficient to establish exposure to their products, because Mr. Tamraz must identify a "particular product" to which he was exposed.

Put simply, none of the cases cited by ESAB stands for this proposition. Statements in *Garcia* and *Lineaweaver* that a plaintiff must supply substantial evidence of exposure to a "particular" product are pointed at circumstances where there was *no* particularity of identification at all, not even by manufacturer; the courts' statements do not mean that California law requires plaintiffs to supply direct testimony of the model or serial numbers of the toxic products to which they were exposed. [121] In *Dumin*, the plaintiff "did not say with even a semblance of certainty" that *any*

product manufactured by the defendant was available at his worksite. [122] Here, in contrast, Mr. Tamraz stated with no uncertainty that he actually used ESAB products.

ESAB does not dispute that Mr. Tamraz's testimony supplied substantial evidence of exposure to *some* of its mild steel welding consumable products, even if Mr. Tamraz did not identify precisely which ones. [123] Further, the evidence is undisputed that all mild steel welding consumables contain manganese. And, as discussed above in section II.B of this opinion, defendants' own documents concede that the manganese in fumes given off by mild steel welding consumables will frequently exceed the TLV. In sum, Mr. Tamraz's direct testimony that he used welding consumables manufactured by ESAB provides substantial evidence that he was exposed to toxins emitted by ESAB's products. No more is required to defeat ESAB's motion.

b. Hobart.

The Court's analysis for Hobart is virtually the same. As revealed by the testimony quoted above, Mr. Tamraz stated to the jury six different times that he used welding consumables manufactured by Hobart. Twice, Mr. Tamraz even identified more precisely the type of Hobart welding consumables he used: "We used Lincoln 7018 along with, I think, Hobart 7018," and also "You have Hobart stick; I think I have used some Hobart MIG." This testimony regarding use of Hobart products was unequivocal, as was his identification of the Hobart logo.

Like ESAB, Hobart argues that Mr. Tamraz was required to identify with greater particularity the exact Hobart welding consumables he used. Addressing this point further, Hobart observes that, when Mr. Tamraz testified that he used Hobart 7018 and Hobart MIG, he stated "I think;" Hobart insists this language classifies Mr. Tamraz's recollection as mere conjecture. Again, however, Mr. Tamraz was not required to identify exactly which Hobart welding consumables he used; he was required only to provide substantial evidence that he used *some* Hobart welding consumables, and that his use of Hobart welding products (regardless of precisely which ones) exposed him to manganese fumes. Further, even if the Court agreed that Mr. Tamraz's use of the phrase "I think" indicated he was less than sure, it certainly does not weaken his testimony to the point of speculation. Mr. Tamraz's direct testimony that he used welding consumables manufactured by Hobart meets the test for substantial evidence of exposure.

c. TDY.

Mr. Tamraz stated to the jury four different times that he used McKay welding consumables. TDY offers three reasons why this identification was not substantial evidence that Mr. Tamraz was exposed to welding consumables that were manufactured by TDY. The first reason is the same as the one offered by ESAB and Hobart -- Mr. Tamraz did not offer sufficiently particularized evidence of which TDY welding consumables he used. For the same reasons this argument failed for ESAB and Hobart, it fails for TDY.

The second and third reasons offered by TDY both have to do with the fact that the McKay brand name did

not always belong to TDY during Mr. Tamraz's welding career. As the Court instructed the jury, "defendant TDY Industries, Inc. ceased manufacturing welding consumables on December 12, 1992." [124] TDY notes that, before this time, it sold welding consumables under the brand name "Teledyne McKay;" after that time, TDY left the welding consumables business and defendant Hobart sold welding consumables under the "McKay" brand name. [125] TDY argues that, because Mr. Tamraz stated he used "McKay" welding consumables, and not "Teledyne McKay" consumables, his product identification is insufficient. TDY further argues that, even if Mr. Tamraz's reference to "McKay" consumables was otherwise sufficient to implicate TDY, his product identification still fails because Mr. Tamraz did not make clear that he used these "McKay" consumables before 1993. Given the applicable legal standards, however, the Court is ultimately unpersuaded that TDY is entitled to judgment notwithstanding the verdict.

To begin, Mr. Tamraz's statement that he used "McKay" welding consumables and not "Teledyne McKay" consumables provides little traction for TDY's product identification argument. Welders commonly use nicknames and verbal shorthand when referring to the consumables they use, such as "MIG" for Metal Inert Gas wire, or "Fleetweld 5" for Lincoln's 6010 welding rod. By itself, Mr. Tamraz's statement that he used "McKay" welding consumables is at least as consistent with his having used TDY-manufactured products as with his having used a "McKay" product manufactured by Hobart; his testimony certainly does not mandate an inference that he used only Hobart's McKay rods. And "if several reasonable inferences may be drawn, the

motion for judgment notwithstanding the verdict should be denied." [126]

More to the point, there was introduced at trial enough evidence upon which a reasonable jury could conclude that the "McKay" products referred to by Mr. Tamraz were, in fact, manufactured by TDY. Mr. Tamraz was questioned explicitly about the 1992 time period, when McKay consumables were still being manufactured by TDY:

> Q1. Were you welding in 1992?
>
> A1. Yes.
>
> Q2. At times, did you use welding consumables manufactured by ESAB?
>
> A2. Yes.
>
> Q3. What other manufacturers' consumables did you use?
>
> A3. Well, that I can recall, I used Lincoln quite a bit. I used Hobart, Airco, Linde, ESAB, and McKay. That's about all I can remember.
>
> Q4. Thank you. [127]

In addition, Mr. Tamraz was shown the "Teledyne McKay" logo and he confirmed he used that manufacturer's products. [128] He also testified he used McKay products "earlier in [his] career," which began in 1978. [129]

Defendants argue that each bit of this evidence regarding time-frame suffers some weakness, so that the sum of this evidence provides insufficient support for the conclusion that Mr. Tamraz used a McKay welding consumable manufactured by TDY *before 1993* (as opposed to a McKay consumable manufactured later by Hobart). For example, defendants assert that: (1) "earlier in my career" can still mean "after 1992;" and (2) Mr. Tamraz seemed initially unsure whether the "Teledyne McKay" logo he was shown was the same as the logo on the products he used. [130]

The Court agrees with defendants that the foundation constructed by Mr. Tamraz to support his assertion that he used TDY-manufactured welding consumables could have been more solid and that, based on this evidence, the jury could reasonably have rendered a verdict in TDY's favor. But the Court is also mindful that it may grant TDY's motion "only if it appears from the evidence, viewed in the light most favorable to [Mr. Tamraz], that there is *no* substantial evidence to support the verdict. If there is *any* substantial evidence, or reasonable inferences to be drawn therefrom, in support of the verdict, the motion should be denied." [131]

In this case, the jury was presented with testimonial evidence directly from Mr. Tamraz that he used Teledyne McKay welding consumables when he was welding *in 1992*. Regardless of whether he called these consumables "McKay" rods, or "Teledyne McKay" rods, or even "Teledyne" rods, it is not disputed that a 1992 consumable so identified had to have been a TDY product. Hobart was not responsible for any "McKay" welding consumable until 1993. [132]

This Court may not re-weigh the evidence nor assess credibility. The Court instructed the jury it could only find against TDY if it determined that Mr. Tamraz used TDY products manufactured before December 12, 1992, and -- while the jury had to engage in some reasonable inferential leaps to reach its final determination -- there was substantial evidence to support its conclusion that TDY was liable. Accordingly, TDY's motion, based on lack of product identification, must be denied.

d. BOC.

Mr. Tamraz stated to the jury four different times that he used "Airco" welding consumables. Borrowing a page from TDY's motion, BOC argues this product identification was not substantial evidence that Mr. Tamraz was exposed to welding consumables that were manufactured *by BOC*, because the Airco brand name did not always belong to BOC during Mr. Tamraz's welding career. This time, however, the Court agrees: unlike in the case of TDY, Mr. Tamraz did not adduce substantial evidence that he used Airco products manufactured by BOC during the relevant time-frame. [133]

As the Court instructed the jury, BOC "ceased manufacturing welding consumables completely after 1986. * * * After 1986, Airco products were sold under that name by defendant the Lincoln Electric Company." [134] As such, defendant BOC may be found liable to Mr. Tamraz only if the jury received substantial evidence that he was exposed to Airco welding consumables manufactured in 1986 or before. None of the time-frame references offered by Mr. Tamraz, however, provide

more than a speculative basis upon which to reach this conclusion.

A comparison of TDY's and BOC's product-identification evidence is illustrative. Mr. Tamraz was shown a "Teledyne McKay" logo and testified he used products manufactured by that defendant. The "Teledyne McKay" logo shown was different from the "McKay" logo that was used later, by Hobart. In contrast, Mr. Tamraz was simply shown an "Airco" logo, and there was presented no evidence suggesting this logo was different before 1987 (when BOC manufactured Airco consumables) compared to after 1986 (when Lincoln manufactured Airco consumables). In other words, Mr. Tamraz's testimony regarding product branding did nothing to differentiate Lincoln's Airco products from BOC's Airco products.

Mr. Tamraz also testified regarding the products he used in 1992, when "McKay" consumables were still being manufactured by TDY. Mr. Tamraz's testimony that he used Airco products in 1992, however, shows only that he was then using a Lincoln consumable; it says nothing about whether he had used a BOC-manufactured Airco consumable six-or-more years earlier. Finally, Mr. Tamraz testified he used McKay products "earlier in his career," but he did not so testify about Airco products. [135] Ultimately, while the jury was presented with substantial evidence from which it could reasonably infer that Mr. Tamraz used TDY-manufactured "McKay" consumables, there was no similar time-frame evidence from which a jury could draw reasonable inferences that Mr. Tamraz used BOC-manufactured "Airco" consumables.

In *Garcia*, it was certain the plaintiff had been injured by a sabre manufactured by either American or Vince, but the evidence would have required a jury to speculate which one. In this case, it is certain that Mr. Tamraz used Airco welding consumables manufactured by either Lincoln or BOC, but Mr. Tamraz did not provide sufficient time-frame evidence to allow the jury to do more than speculate which one. Because the jury's conclusion that Mr. Tamraz used BOC's Airco product is necessarily based upon conjecture and surmise, rather than upon reasonable inference, the Court finds BOC's motion for judgment as a matter of law, notwithstanding the verdict, is well taken.

5. Additional Grounds.

In addition to the product identification arguments asserted individually by ESAB, Hobart, and TDY, discussed above, these three defendants also assert two additional, common grounds for judgment as a matter of law. First, TDY and ESAB note that Mr. Tamraz never displayed to the jury one of their actual warning labels, and insist this was required before a jury could find against them. In fact, TDY and ESAB go so far as to state that "plaintiffs never presented *any* ESAB [or TDY] warning language whatsoever to the jury." [136] This latter statement, however, is simply wrong. As discussed above, Mr. Tamraz presented evidence that the AWS promulgated standardized warning label language, and all of the defendants adopted this language. The defendants, themselves, referred during trial to "the 1967 warning," in a context that can only be understood to mean that the defendants all used the same warning language. The jury was shown several exemplars of 1967 warning labels containing the same

language -- specifically, AWS memoranda discussing the mandated language, as well as actual warning labels used by Lincoln, Hobart, and BOC. Thus, the jury *was* presented with substantial evidence regarding the content of ESAB's and TDY's warning labels. Where the defendants conceded they all used the same language, the fact that the jury did not see an actual ESAB or TDY label is not a valid ground for vacating the jury's verdict against them.

The second common argument asserted by ESAB, Hobart, and TDY is that Mr. Tamraz did not define precisely when he used each of their products during his career, and thus did not make clear which of their warnings were actually at issue as having been insufficient. More specifically, Mr. Tamraz testified he saw the defendants' warnings in the 1978-79 time frame. [137] This means Mr. Tamraz saw what is referred to as "the 1967 warning" and possibly also "the 1979 warning," both of which were promulgated by the AWS and used by all defendants. ESAB, Hobart, and TDY assert that, without particularized proof that Mr. Tamraz used their products *during the time they were labeled with the 1967 or 1979 warnings*, there can be no causal connection between his injuries and their failure to warn. For example, ESAB argues as follows:

> As the Court is aware, ESAB warnings -- like those of other manufacturers of welding consumables -- changed over the course of Mr. Tamraz's career. For example, ESAB . . . began including manganese-specific information in its material safety data sheets around 1992. Moreover, the company added a "central nervous system" warning to its welding consumables in

> 2002 -- while Mr. Tamraz was still actively welding. Without evidence establishing precisely when Mr. Tamraz allegedly used an ESAB product and precisely what product he used, the jury was powerless to identify which ESAB warning was at issue and to assess whether the content of that particular warning provided appropriate information. In addition, without knowing which ESAB warning Mr. Tamraz claimed was inadequate, the jury could not have properly determined whether that warning proximately caused his alleged injury. Thus, the jury in this case essentially determined that ESAB's warning was both inadequate and caused Mr. Tamraz physical harm without knowing what that warning was or what it said. [138]

The problem with this argument is that it ignores the central premise of Mr. Tamraz's entire case -- *all* of the labels that accompanied *all* of the defendants' products he used during *all* of his career were inadequate, because the labels did not warn explicitly that exposure to welding fumes could cause permanent, irreversible brain damage that becomes progressively worse over time. Mr. Tamraz and his experts insisted repeatedly at trial that this warning inadequacy has continued through today, even as some of the manufacturers, at various points in time, added manganese-specific cautions to their material safety data sheets ("MSDSs"), because these MSDS cautions were not contained in the warning labels, themselves. Thus, in fact, the jury *did* know "which ESAB warning was at issue" -- all of them. Essentially, Mr. Tamraz argued that *every* ESAB

welding consumable he used was defective because it contained an inadequate warning, and the jury agreed.

TDY adds the argument that Mr. Tamraz had to prove he used a TDY product that was accompanied by the 1967 warning, and not some later warning, because: (1) Mr. Tamraz testified he only saw the 1967 label; and (2) he cannot prevail on a failure-to-warn claim if he did not read the warning that accompanied the product he used. This argument fails for two reasons. First, Mr. Tamraz's testimony can be read to understand that he saw both the 1967 label and the 1979 label. Second, it is not true that Mr. Tamraz "cannot prevail on failure-to-warn claims if he did not read the warning that accompanied the product he used." [139] If -- as Mr. Tamraz asserted, and as the jury could reasonably have found -- part of the reason he did not read a warning is that the defendants purposely made the label hard to find and to read, through its placement and size, then he is not precluded from pursuing his failure-to-warn claim. [140]

In sum, the Court concludes that the additional common arguments asserted by ESAB, Hobart, and TDY are not persuasive bases for granting their motions for judgment notwithstanding the verdict.

III. The Effect of Granting BOC's and TDY's Motions.

Having granted BOC's post-verdict motion for judgment as a matter of law, the question arises: What effect does this ruling have on the jury's verdict? For the reasons explained below, the answer is that the verdict remains intact and unchanged.

Before trying Mr. Tamraz's case, this Court presided over the bellwether *Welding Fume* MDL trial of *Solis v. Lincoln Electric Co.*, case no. 04-CV-17363. The same five defendants that went to trial in *Tamraz* also went to trial in *Solis*. In *Solis*, the Court initially drafted a verdict form directing the jury: (1) to determine whether *any* of the defendants were liable for failure to warn; (2) if there was any liability, to determine a total amount of compensatory damages; and (3) if there were compensatory damages, to determine what percentage of the plaintiff's injuries each defendant had caused. [141] Had the Court used this version of the verdict form (and had the jury awarded compensatory damages), the Court's final judgment would have entered a specific amount of compensatory damages against each defendant.

The *Solis* defendants, however, asked the Court to direct the jury to determine a *single* compensatory damage amount, which they would then allocate amongst themselves. Accordingly, the actual, final Verdict Form in *Solis* directed the jury simply to: (1) determine whether each defendant, individually, was liable for failure to warn; and (2) if there was any liability, determine a *total* amount of compensatory damages. [142] This Verdict Form allowed for entry of no individualized, defendant-specific damage amounts in the Court's final judgment. Although the defendants never explained explicitly why they preferred this approach, the Court assumed it was because, well before the inception of the *Welding Fumes* MDL, the defendants (and other welding consumable manufacturers) had entered into a judgment-sharing agreement. [143]

In any event, during its charge conference with the parties in *Tamraz*, the Court asked the defendants whether, as they had in *Solis*, they wanted the jury simply to determine a single, total compensatory damage award. The defendants confirmed they did want the Verdict Form to direct the jury to determine only a single, total amount of compensatory damages, not broken out by defendant. [144] After the jury entered its verdict, which awarded the Tamrazes a total of $ 20.5 million in compensatory damages, the defendants orally renewed their Rule 50 motions for judgment as a matter of law. The Court responded as shown below:

> THE COURT: All right. Well, your motion has been renewed. You can certainly renew it in writing if you would like. I just want to remind you that to the extent that any ruling leaves any defendant still in the case, the compensatory damage verdict remains intact because I specifically gave the defendants the opportunity to have the jury separate out the damage award, and I specifically asked whether or not there was any need to separate out any damage award. So even if it were true that I were to agree with you, say, as to TDY, it doesn't change the damage award if any defendant remains. So if, say, Lincoln remained and no one else did, there is no ability to then attack it, because to say that it should be somehow apportioned because I gave you all the ability, and I asked you several times on the record to confirm that fact, so that there would be no question if the matter were to go up on appeal. So I just want to make that clear. [145]

> Defense counsel replied: "That's confirmed, Your Honor." [146]

In sum, the effect of granting BOC's motion is essentially known only to the defendants, as they agreed that any allocation of an award of compensatory damages was a purely internal matter. That portion of the Court's judgment awarding a total of $ 20.5 million in compensatory damages to the Tamrazes, however, remains intact and unchanged.

IT IS SO ORDERED.

/s/ Kathleen M. O'Malley

KATHLEEN McDONALD O'MALLEY
UNITED STATES DISTRICT JUDGE
DATED: July 18, 2008

Footnotes

1 *See* docket no. 168 (final judgment).

2 *See* trial tr. at 2028-46 (Nov. 15, 2007) (denying motions for directed verdict after close of plaintiffs' case); *id.* at 2600-26 (Nov. 20, 2007) (denying motions for directed verdict after close of defendants' case).

3 Fed. R. Civ. P. 50(b).

4 *Id.* "The movant may alternatively request a new trial or join a motion for a new trial under Rule 59." *Id.* The defendants did not request a new trial in this case.

5 Defendants orally renewed their Rule 50 motions

immediately after the verdict was delivered, and then filed the six written motions listed above. *See* trial tr. at 2919-22 (Dec. 5, 2007) (orally renewing motions).

6 Fed. R. Civ. P. 50(b).

7 *Mannix v. County of Monroe*, 348 F.3d 526, 531 (6th Cir. 2003) ("[i]n federal court diversity cases, this circuit adheres to the minority rule that state law governs the standard for granting motions for directed verdicts and judgments notwithstanding the verdict"); *Potti v. Duramed Pharmaceuticals, Inc.*, 938 F.2d 641, 645 (6th Cir. 1991) ("a federal court sitting in diversity applies the standard for a directed verdict used by the courts of the state whose substantive law governs the action").

8 *In re Coordinated Latex Glove Litigation* ("*Latex Glove*"), 99 Cal. App. 4th 594, 121 Cal.Rptr.2d 301, 309 (Cal. Ct. App. 2002). Further, "the trial judge's power to grant a judgment notwithstanding the verdict is identical to his power to grant a directed verdict." *Id.*

9 *Id.; see Goetz v. Hess*, 2002 Cal. App. Unpub. LEXIS 6660, 2002 WL 1608403 at *1 (Cal. Ct. App. July 22, 2002) ("The court must disregard all conflicting evidence and indulge in every legitimate inference that may be drawn in support of the judgment, and may grant the motion only if there is no substantial evidence to support the verdict.").

10 *Latex Glove*, 121 Cal.Rptr.2d at 309 (emphasis added).

11 *Howard v. Owens Corning*, 72 Cal. App. 4th 621, 85 Cal.Rptr.2d 386, 393 (Cal. Ct. App. 1999) (some

emphasis added). The applicable legal standards under California law are essentially the same as those the Court would apply if this case had federal subject matter jurisdiction. In such instances, when determining whether to grant judgment as a matter of law, the Court is not free to weigh the parties' evidence or to pass upon the credibility of witnesses. *Black v. Zaring Homes*, 104 F.3d 822, 825 (6th Cir. 1997); *K & T Enters. v. Zurich Ins. Co.*, 97 F.3d 171, 175 (6th Cir. 1996). Nor may the Court substitute its own judgment for that of the jury. *Zaring Homes*, 104 F.3d at 825; *K & T Enters.*, 97 F.3d at 175-76. Instead, the Court must view the evidence most favorably to the party against whom the motion is made and give that party the benefit of all reasonable inferences from the record. *Zaring Homes*, 104 F.3d at 825; *K & T Enters.*, 104 F.3d at 176. When the evidence would permit reasonable minds to differ on the issues decided by the jury, a motion for judgment as a matter of law must be denied. *American and Foreign Ins. Co. v. Bolt*, 106 F.3d 155, 1997 WL 57361 (6th Cir. 1997). In short, every effort must be made to uphold the verdict, if reasonably possible.

12 Motion at 1 (docket no. 174).

13 *Id.* At the close of their case, the defendants filed a written motion for judgment as a matter of law on this ground, pursuant to Rule 50(a). *See* docket no. 155. Defendants did not earlier make a similar motion after the close of plaintiffs' case.

14 *Id.* at 5 (quoting *Motus v. Pfizer, Inc.*, 196 F. Supp.2d 984, 991 (C.D. Cal. 2001), *affirmed*, 358 F.3d 659 (9th Cir. 2004)).

15 *Id.*

16 6 Cal. 4th 539, 25 Cal. Rptr. 2d 97, 863 P.2d 167 (Cal. 1993)

17 147 Fed. Appx. 651, 2005 WL 2108654 (9th Cir. Sept. 1, 2005)

18 819 F.2d 349 (2nd Cir. 1987) (applying California law)

19 *Lord v. Smithkline Beecham Corp.*, 2007 Cal. App. Unpub. LEXIS 10322, 2007 WL 4418019 at *3 (Cal. Ct. App. Dec. 19, 2007) (concluding the presumption was rebutted).

20 *Reyes v. Wyeth Laboratories*, 498 F.2d 1264, 1281 (5th Cir. 1974), *cert. denied*, 419 U.S. 1096, 95 S. Ct. 687, 42 L. Ed. 2d 688 (1974). The presumption "works in favor of the manufacturer when an adequate warning is present. Where there is no warning . . ., however, the presumption that the user would have read an adequate warning works in favor of the plaintiff user. In other words, the presumption is that [the plaintiff] would have read an adequate warning. The presumption, may, however, be rebutted if the manufacturer comes forward with contrary evidence that the presumed fact did not exist." *Id.*

21 *See* trial tr. at 2605-2611 (Nov. 20, 2007).

22 *Lord*, 2007 Cal. App. Unpub. LEXIS 10322, 2007 WL 4418019 at *3.

23 A digression: during trial, the plaintiffs filed a motion asking the Court to preclude the defendants

from raising the sophisticated user defense, which is related to the learned intermediary doctrine. *See* docket no. 145 (plaintiffs' motion for summary judgment). The Court ultimately declined to charge the jury on the sophisticated user defense, for two reasons: (1) there was some question whether California courts recognized the defense; and (2) more importantly, the evidence presented at trial did not even fairly raise the issue. *See* trial tr. at 2618-21 (Nov. 20, 2007) ("I did not charge the jury with a separate independent sophisticated user defense both because there is a serious question under California law as to what the nature of that defense is, and [because] the most recent decision that I have from [California Coordinated Proceeding] Judge Sabraw is that it is not appropriate to even allow that defense; but separate and apart from that, there simply is not substantial evidence in the record discussing the nature of the users, meaning the intermediaries, here, and there wasn't sufficient evidence in the record to allow the Court to even instruct as it relates to the nature of the warnings provided to a particular employer [qua sophisticated user]."). (Note - Judge Bonnie Sabraw is presiding over California Judicial Council Coordination Proceeding No. 4368, the state-court analog to this federal MDL).

Subsequently, the California Supreme Court concluded, on a "question of first impression," that "the sophisticated user defense applies in California." *Johnson v. American Standard, Inc.*, 43 Cal. 4th 56, 74 Cal. Rptr. 3d 108, 179 P.3d 905, 908 (Cal. 2008). Given the Court's second reason for declining to instruct the jury on the sophisticated user defense, however, the *Johnson* holding is not relevant to any issues raised by the defendants.

24 Jury Instructions at 28 (docket no. 160). It is also worth noting that the *Massok* case, cited by defendants, states as follows: "We also pause to note that Massok's failure to read the warning labels, even if considered negligent, does not necessarily bar recovery under California's comparative negligence regime. Regardless of the plaintiff's negligence in using the product, a manufacturer is still under a duty to provide consumers with a non-defective product, and thus any fault on the part of the plaintiff is simply factored into the more general comparative negligence calculus when assessing the parties' relative responsibility for the injury." *Massok*, 147 Fed. Appx. 651, 2005 WL 2108654 at *8 n.3 (9th Cir. 2005). The *Massok* court reversed the district court's order granting to defendants judgment as a matter of law.

25 Motion at 1 (docket no. 174).

26 Trial tr. at 794-06, 833 (Nov. 7, 2007).

27 *Id.*

28 *Id.* at 827-28, 910.

29 *Id.* at 794-06, 833.

30 *Id.*

31 *Id.* at 1591-92 (Nov. 12, 2007).

32 *Latex Glove*, 121 Cal.Rptr.2d at 309.

33 Motion at 1 (docket no. 175).

34 *Lineaweaver v. Plant Insulation Co.*, 31 Cal. App. 4th 1409, 37 Cal. Rptr.2d 902, 905 (Cal. Ct. App. 1995).

35 *Id.*

36 *Id.* (citations omitted).

37 *Id.* at 906 (citation omitted). *See also Redfoot v. B.F. Ascher & Co.*, 2007 U.S. Dist. LEXIS 40002, 2007 WL 1593239 at *15 (N.D. Cal. June 1, 2007) ("Under California law, causation in a personal injury action must be proven within a reasonable medical probability based on competent expert testimony.").

38 *Stadish v. Southern California Gas Co.*, 2002 Cal. App. Unpub. LEXIS 5648, 2002 WL 1360667 at *7 (Cal. Ct. App. June 24, 2002).

39 2002 Cal. App. Unpub. LEXIS 5648, [WL] at *10.

40 *Id.* (internal quotation marks and citation omitted).

41 *Lineaweaver*, 37 Cal. Rptr.2d at 906. The *Lineaweaver* court cited *Restatement Second of Torts* § 433B (1965) several times in its discussion of burden of proof, causation, and exposure. *Lineaweaver*, 37 Cal. Rptr.2d at 906-07. In relevant part, the *Restatement* states: "The plaintiff is not, however, required to prove his case beyond a reasonable doubt. He is not required to eliminate entirely all possibility that the defendant's conduct was not a cause. It is enough that he introduces evidence from which reasonable men may conclude that it is more probable that the event was caused by the defendant than that it was not. The fact of causation is incapable of mathematical proof, since no man can say

with absolute certainty what would have occurred if the defendant had acted otherwise. If, as a matter of ordinary experience, a particular act or omission might be expected to produce a particular result, and if that result has in fact followed, the conclusion may be justified that the causal relation exists. In drawing that conclusion, the triers of fact are permitted to draw upon ordinary human experience as to the probabilities of the case." *Id.* § 433B, cmt. b.

42 *See* trial tr. at 1323 *et seq.* (Nov. 9, 2007) (testimony of Mr. Kahane).

43 The ACGIH describes a TLV as follows: "[TLVs] refer to airborne concentrations of chemical substances and represent conditions under which it is believed that nearly all workers may be repeatedly exposed, day after day, over a working lifetime, without adverse health effects. TLVs are developed to protect workers who are normal, healthy adults." *See* www.acgih.org/Products/tlv_bei_intro.htm. *See In re Welding Fume Prods. Liab. Litig.*, 526 F.Supp.2d 775, 784 n.33 & 789-90 n.65 (N.D. Ohio 2007) (discussing the history and meaning of the TLVs and PELs for manganese).

44 The ACGIH has also promulgated a measure similar to OSHA's PEL, which it terms an "excursion limit," and which is set at 1.0 mg/mm<3>.

45 Battelle Survey at 27 (trial exh. 1562) (emphasis added).

46 *Id.* at 65.

47 Lincoln Powerpoint presentation at 12 (2004) (trial exh. 6112)

48 AWS Safety & Health Committee Meeting minutes at 5 (May 20, 1981) (trial exh. 1647).

49 *See* trial tr. at 2126-30 (Nov. 15, 2007) (review of testimony of Lincoln representatives John Stropki, Marie Quintana, and Kenneth Brown, that there is no known safe level of exposure to manganese in welding fume, and/or that the level at which exposure becomes unsafe is not known).

50 Trial tr. at 1343 (Nov. 9, 2007). Substantial evidence was presented showing that defendants were aware that welding fume exposure could cause Manganese-Induced Parkinsonism ("MIP"). In 1979, a widely-distributed AWS literature review noted that "[p]otential exposure to manganese occurs whenever this metal is used in electrode coatings or in electrode wire," and that manganese is "poisonous to the nervous system." American Welding Society, "*Effects of Welding on Health*," at xix (1979) (trial exh. 2940). This literature review went on to state that the "observation that manganism resembles Parkinson's disease deserves emphasis. Although no data on the prevalence of parkinsonism in welders are available, there is a concern that some cases of manganese poisoning could be mistakenly diagnosed as Parkinson's disease. Further investigations may be warranted." *Id.* at 29. In 1980, commenting on another welding fume lawsuit that was widely discussed within the industry, one manufacturer wrote the following summary: "[the plaintiffs] complained of the fumes for 3 weeks, then came down with symptoms of manganese poisoning.

They have severe neurological damage which . . . is doubtless due to the welding in a confined area with the Hadfield Manganese rods. The damage is irreversible - these guys are impaired for life." AWS Safety & Health Committee meeting minutes at 5 (May 20, 1981) (trial exh. 1647). In 1992, an internal ESAB memorandum also acknowledged the same hazard, stating: "Once [welding rods] are used in an electric arc, they can liberate respirable manganese fumes. * * * Manganese fumes are of serious concern. The irreversible neurological damaged produced by chronic overexposure to manganese fume is tragic. You are wise to be concerned." ESAB memorandum from George Barnes to William Esch (Feb. 24, 1992) (trial exh. 260).

51 AWS Fumes and Gases Committee meeting minutes at 3 (Oct. 17, 1994) (trial exh. 920).

52 *See In re Welding Fume Prods. Liab. Litig.*, 245 F.R.D. 279, 316 (N.D. Ohio 2007) (in the context of denying certification of a medical monitoring class, reviewing plaintiffs' evidence and concluding that "at least some defendants have recognized that some welder-plaintiffs: (1) are routinely exposed to manganese in welding fumes above safe threshold limits; [and] (2) thereby suffer an increased risk of developing neurological illness due to exposure to welding fumes") (footnotes omitted).

53 *See* trial tr. at 1350-55 (Nov. 9, 2007) (testimony of Mr. Kahane regarding the OSHA database).

54 *See* trial tr. at 1476 *et seq.* (Nov. 12, 2007) (testimony of Mr. Montoya).

55 Trial exh. 1155; trial tr. at 1651-53 (Nov. 12, 2007) (counsel reading this language from the MSDS). *See also* trial tr. at 1373 (Nov. 9, 2007) (counsel referring to an MSDS that stated "manganese can cause central nervous system injury, including brain damage").

56 In 1992, an internal ESAB memorandum acknowledged this same hazard, stating: "Once [welding rods] are used in an electric arc, they can liberate respirable manganese fumes. * * * Manganese fumes are of serious concern. The irreversible neurological damaged produced by chronic overexposure to manganese fume is tragic. You are wise to be concerned." ESAB memorandum from George Barnes to William Esch (Feb. 24, 1992) (trial exh. 260).

57 Trial tr. at 1335 (Nov. 9, 2007).

58 *Id.* at 633 (Nov. 6, 2007) (Dr. Burns quoting the WHO publication, "Environmental Health Criteria for Manganese," trial exh. 1547 at 13). *See also id.* at 959 (Nov. 7, 2007) (Mr. Tamraz's treating neurologist, Dr. Walter Carlini, acknowledging individual susceptibility to manganese exposure); *Daubert* hearing tr. at 1995 (July 26, 2005) (defense expert Dr. Warren Olanow agreeing that susceptibility to the adverse effects of manganese varies from individual to individual).

The Court does not recite in this opinion all of the substantial evidence supporting the proposition that exposure to manganese in welding fumes can, at least in some circumstances, cause Manganese-Induced Parkinsonism - a proposition the defendants now concede. The Court examined much of this evidence in great depth in the context of assessing its admissibility

under the standards set out in *Daubert*. *See In re Welding Fume Prods. Liab. Litig.*, 2005 U.S. Dist. LEXIS 46164, 2006 WL 4507859 (N.D. Ohio Aug. 8, 2006) (actually issued on August 8, 2005, *see* case no. 03-CV-17000, docket no. 1352).

59 Motion at 4 (docket no. 175) (emphasis added) (quoting *McClain v. Metabolife Intl., Inc.*, 401 F.3d 1233, 1242 (11th Cir. 2005) (internal quotation marks omitted)).

60 Federal Judicial Center, *Reference Manual on Scientific Evidence* 405 (2nd ed. 2000). *See Plourde v. Gladstone*, 190 F.Supp.2d 708, 721 (D. Vt. 2002) ("In most toxic tort cases it is impossible as a matter of practice to quantify with hard proof - such as the presence of the alleged toxic substance in the plaintiff's blood or tissue - the precise amount of the toxic substance to which an individual plaintiff was exposed.").

61 *Stadish*, 2002 Cal. App. Unpub. LEXIS 5648, 2002 WL 1360667 at *7.

62 *Bonner v. ISP Technologies, Inc.*, 259 F.3d 924 (8th Cir. 2001).

63 *Id.* at 928 (citation and internal quotation marks omitted).

64 *Id.* at 932.

65 *Id.* at 931.

66 *Lineaweaver*, 37 Cal. Rptr.2d at 906.

67 Mr. Tamraz testified that, on days that he worked as a welder, he was frequently exposed to copious welding fumes; and employers regularly requested his services, so that the regularity of his work-day exposure was also high. *See Lohrmann v. Pittsburgh Corning Corp.*, 782 F.2d 1156, 1162 (4th Cir. 1986) (sufficiency of evidence of asbestos exposure "would depend upon the frequency of the use of the product and the regularity or extent of the plaintiff's employment in proximity thereto").

68 Defendants listed an expert industrial hygienist on their witness list, but did not call him at trial.

69 Regarding defendants' argument that the opinions expressed by plaintiff's expert industrial hygienist, Mr. Kahane, were unsupported, this Court borrows a comment from another court that was presented with the identical argument: "In our view, however, [plaintiff's expert] drew a reasonable inference rather than make an unwarranted assumption of fact. Apparently the jury agreed." *Kritser v. Beech Aircraft Corp.*, 479 F.2d 1089, 1095 (5th Cir. 1973) (citing *Sentilles v. Inter-Caribbean Shipping Corp.*, 361 U.S. 107, 109-10, 80 S. Ct. 173, 4 L. Ed. 2d 142 (1959)).

70 *See* trial tr. at 2673-76 (Nov. 20, 2007) (defense counsel agreed that, on the verdict form, "the defendants did not want compensatory damages separated out per defendant," and "if there is any allocation among the defendants, they will deal with that"). This point is discussed further below, in section III of this opinion.

71 Motion at 1 (docket no. 170) (emphasis added).

72 *See In re Welding Fume Prods. Liab. Litig.*, 526 F.Supp.2d at 782 & 791-92 (discussing the AWS's adoption of the 1967 and 1979 standard labels).

Indeed, counsel for plaintiffs, in opening statement, explained at length that the defendants all used the same AWS-promulgated warning language from 1967 through the mid-1980's, and defendants adopted this characterization throughout trial. *See* trial tr. at 492-96 (Nov. 5, 2007) (plaintiffs' opening statement regarding the evolution of industry warning labels); *id.* at 2044 (Nov. 15, 2007) (during oral argument on defendants' mid-trial Rule 50 motions, the Court observed it was "not particularly concerned about the question of the specificity of the labels, which were or were not shown to the jury, because it is true that there are comments by defense counsel and certainly substantial arguments by defense counsel at side bars that the warnings were all, to the extent that they were on products, were all effectively the same, regardless of the nature of the product or the defendant, at least prior to the 1990s time frame.").

It is also notable that, in a prior bellwether trial before this MDL court, the parties agreed that the defendants all used the same warnings. *See Solis v. Lincoln Elec. Co.*, case no. 04-CV-17363, 2006 U.S. Dist. LEXIS 3869, (N.D. Ohio) (O'Malley, J.) (docket no. 195) (Jury Instructions at page 26, stating: "In this case, the evidence has shown that Mr. Solis welded beginning some time in 1974 and ending some time in 1999. During that time frame, defendants used three different warnings: one from 1967 to 1979; one from 1979 to 1997, and one from 1997 forward. Defendants agree that they all employed the same warnings during these three

time periods."). The parties agreed, however, not to include this language in the *Tamraz* jury instructions; *see* trial tr. at 2658-62 (Nov. 20, 2007) (discussing jury instructions regarding consideration of warnings; the draft jury instruction being discussed in this transcript section, which is at docket no. 159, was the instruction used in *Solis*).

73 "Z49.1" refers to American National Standards Institute ("ANSI") Standard Z49.1, entitled "Safety in Welding, Cutting, and Allied Processes." The 1967 version of ANSI Standard Z49.1 is 67 pages long, not including table of contents or appendices, *see* trial exhs. 1621, 2845.

74 See, for example, trial exhibits 92 (Hobart), 1149 (BOC/Airco), and 2008 (Lincoln). As noted above, while the language in these labels is virtually identical, there are very minor differences. For example, some of the defendants' 1967 labels refer to "fumes and gases," others refer to "gases and fumes," and still others refer to "concentrations of fumes and gases." There are no differences amongst the defendants' 1967 labels, however, any more substantive than these *de minimis* word choices.

75 *See, e.g.*, trial tr. at 563-64 (Nov. 6, 2007) (defense counsel referring in opening statement to "the 1967 warning" used by defendants); *id.* at 842 (Nov. 7, 2007) (defense counsel asking Mr. Tamraz about "the 1967 warning" that would have been in effect when he started welding). *See also id.* at 2036 (Nov. 15, 2007) (during oral argument on the Rule 50 motions, the Court making clear that it had disallowed defense counsel during opening statement from using as

demonstrative evidence a "1967 label" that had never actually appeared on any product, not from asserting that all the defendants' 1967 labels were identical).

76 *See, e.g., id.* at 589 (Nov. 6, 2007) (in opening statement, defense counsel referring, in undifferentiated fashion, to the language of the 1979 warning); *id.* at 1483-1507 (Nov. 12, 2007) (same, in cross-examination of plaintiffs' witness).

77 *See id.* at 1483-1507 (Nov. 12, 2007); *see* trial exhibits 3081, 9653, and 9664. The Lincoln warnings shown to the jury contained the same language as the AWS standard warning label, but the order of some of the sentences was changed.

78 *See* trial tr. at 492-96 (Nov. 5, 2007) (plaintiffs' opening statement regarding the evolution of industry warning labels).

79 Mr. Tamraz began welding in 1978 and stopped working in 2004. The only possible exception to the statement that Mr. Tamraz never used a product accompanied by a warning about brain damage was that, in 2002, two years before Mr. Tamraz stopped welding (and one year after he experienced his first symptoms of brain damage), ESAB began using a label that includes the statement: "Overexposure to manganese compounds can affect the central nervous system, symptoms of which are languor, sleepiness, muscular weakness, emotional disturbances, and spastic gate [sic]. The effect of manganese on the nervous system is irreversible. Manganese . . . [has] low OSHA Permissible Exposure Limits and ACGIH Threshold Limit Values that easily may be exceeded." *See* trial tr.

at 1828-32 (Nov. 13, 2007) (testimony noting that this language, which was on ESAB MSDSs, was added to the warning label). Mr. Tamraz testified, however, that he never saw a label that referred to manganese. *Id.* at 842 (Nov. 7, 2007). Further, defendants objected to introduction of this label at trial on the ground that it was a subsequent remedial measure, and it did not come in to evidence.

80 *In re Welding Fume Prods. Liab. Litig.*, 526 F.Supp.2d 775, 796 (N.D. Ohio 2007) (citation omitted).

81 *Setliff v. E. I. Du Pont de Nemours & Co.*, 32 Cal. App. 4th 1525, 38 Cal.Rptr.2d 763, 767 (Cal. Ct. App. 1995).

82 *Garcia v. Joseph Vince Co.*, 84 Cal. App. 3d 868, 148 Cal.Rptr. 843, 846 (Cal. Ct. App. 1978).

83 *See McGonnell v. Kaiser Gypsum Co., Inc.*, 98 Cal. App. 4th 1098, 120 Cal.Rptr.2d 23, 27 (Cal. Ct. App. 2002) ("A threshold issue in asbestos litigation is exposure to the defendant's product. The plaintiff bears the burden of proof on this issue. If there has been no exposure, there is no causation."). *See also Dumin v. Owens-Corning Fiberglas Corp.*, 28 Cal. App. 4th 650, 33 Cal.Rptr.2d 702, 704 (Cal. Ct. App. 1994) ("The question is whether this evidence would support a jury determination that [the plaintiff] was exposed to [the defendant's product], since [the plaintiff] must establish exposure to prove that his asbestos disease was caused by [the defendant's] conduct in distributing its product.").

84 98 Cal. App. 4th 1098, 120 Cal.Rptr.2d 23 (Cal. Ct. App. 2002).

85 *Id.* at 25.

86 *Id.* at 26.

87 *Id.*

88 *Id.*

89 *Id.* at 28.

90 *Id.* at 29 (emphasis added).

91 28 Cal. App. 4th 650, 33 Cal.Rptr.2d 702 (Cal. Ct. App. 1994).

92 *Id.* at 703.

93 *Id.*

94 *Id.*

95 *Id.* at 706.

96 84 Cal. App. 3d 868, 148 Cal.Rptr. 843 (Cal. Ct. App. 1978).

97 *Id.* at 845.

98 *Id.*

99 *Id.*

100 *Id.* (quoting *Products Liability: Necessity and Sufficiency of Identification of Defendant as Manufacturer or Seller of Product Alleged to Have Caused Injury*, 51 A.L.R.3d 1344, 1349 (1973)).

101 *Id.* at 846. The court added that "the facts gave equal support to two inconsistent inferences thus inviting a verdict based purely on conjecture." *Id.* at 847.

102 The *Garcia* court also distinguished the famous California case of *Summers v. Tice*, 33 Cal. 2d 80, 199 P.2d 1 (Cal. 1948), where two hunters shot toward the plaintiff and birdshot from one of their guns injured the plaintiff's eye. In that case, "both defendants had acted negligently by shooting in [the plaintiff's] direction," while in *Garcia*, at least one of the defendants (the one who had not manufactured the sabre) was clearly not negligent. *Id.* at 847.

103 31 Cal. App. 4th 1409, 37 Cal. Rptr.2d 902 (Cal. Ct. App. 1995).

104 *Id.* at 904.

105 *Id.* at 909.

106 *Id.*

107 *Id.* (emphasis added).

108 *Id.* at 908.

109 In addition to citing *McGonnell*, *Dumin*, *Garcia*, and *Lineaweaver* in support of their motions for

judgment as a matter of law on the basis of insufficient evidence of product exposure, the defendants also cite *Setliff v. E. I. Du Pont de Nemours & Co.*, 32 Cal. App. 4th 1525, 38 Cal.Rptr.2d 763 (Cal. Ct. App. 1995), and *Bockrath v. Aldrich Chemical Co., Inc.*, 21 Cal. 4th 71, 86 Cal. Rptr. 2d 846, 980 P.2d 398 (Cal. 1999). These cases both involve very generalized allegations of exposure to dozens of products made by dozens of manufacturers. The *Setliff* and *Bockrath* courts each ruled that the plaintiff's "toxic soup" complaint required a greater degree of pleading specificity, including identification of "each product that allegedly caused the injury" and an allegation that "each toxin [the plaintiff] absorbed was manufactured or supplied by a named defendant." *Bockrath*, 980 P.2d at 404; *see Setliff*, 38 Cal.Rptr.2d at 767 (affirming judgment in favor of defendants because plaintiff admitted in his amended complaint that was "unable to identify which of the products separately or jointly injured him" and "could not identify the specific chemicals and toxics [sic] involved in his injury or which defendant manufactured the product or products responsible for his injury"). Both *Bockrath* and *Setliff* addressed motions for demurrer made at the pleading stage, not post-trial motions, and so are not fully apposite.

110 *Lineaweaver*, 37 Cal. Rptr.2d at 906.

111 *Id.* at 909.

112 *Id.* at 908.

113 Trial tr. at 786 (Nov. 7, 2007).

114 As discussed further below, before December 12,

1992, TDY sold welding consumables under the brand name "Teledyne McKay;" after this date, Hobart sold welding consumables under the brand name "McKay."

115 As discussed further below, after 1986, Airco products were sold by defendant Lincoln Electric. Mr. Tamraz also testified he used "Linde" welding consumables, but there was presented at trial no evidence regarding which defendant, if any, manufactured Linde consumables.

116 Trial tr. at 820-23 (Nov. 7, 2007).

117 *Id.* at 829-30.

118 *Id.* at 856-57.

119 ESAB Vice President Stanley Ferree testified on cross-examination as follows:

> Q. Are you aware that Jeff Tamraz used welding rods and welding wire manufactured by ESAB?
>
> A. Yes, I think he has identified us as one of the suppliers.
>
> * * *
>
> Q. Okay. Did [another welder who filed suit, Mr. Charles Ruth] use your company's products?
>
> A. I think he identified some, yes.
>
> Q. And he used welding flux cored wire, just like Mr. Tamraz, right?

> A. I'm not sure they were the same type. I think based on Mr. Tamraz's deposition he used mostly the self-shielded type. I think Mr. Ruth used gas shielded type.
>
> Q. Did they both use wire and use the flux core arc welding process?
>
> A.They both used the flux core arc welding process, yes.

Id. at 1819, 1824 (Nov. 13, 2007).

120 In addition to this positive evidence supplied by plaintiff, it is also notable that defendants never argued the negative -- that is, defendants did not assert they did *not* supply their products to Mr. Tamraz's employers. In all *Welding Fume* MDL cases, the parties have focused their discovery on determining which manufacturers' products plaintiff used; this discovery includes not only asking plaintiff whose welding consumables he recalls using, but also, where possible, asking plaintiff's coworkers what products they used, obtaining welding rod purchasing records from plaintiff's employers, and subpoenaing sales records from welding rod distributors and suppliers who served those employers. In this case, Mr. Tamraz's second amended complaint included claims against defendants Thermadyne Holdings Corporation and Deloro Stellite Company, *see* dkt. no. 22, but Mr. Tamraz voluntarily dismissed those defendants after failing to discover sufficient proof that he used their products, *see* dkt no. 47. While it certainly remains Mr. Tamraz's burden at trial to prove he was exposed to each defendant's products, the fact that defendants did

not assert their products were unavailable at Mr. Tamraz's work sites has a modicum of weight.

121 *See Garcia*, 148 Cal. Rptr. at 844 ("to hold a producer, manufacturer, or seller liable for injury caused by a particular product, there must first be proof that the defendant produced, manufactured, sold, or was in some way responsible for the product"); *Lineaweaver*, 37 Cal. Rptr. at 906 (the plaintiff "rightly bears the burden of proving exposure to a particular defendant's product").

122 *Dumin*, 33 Cal. Rptr.2d at 705.

123 An analogy: if the evidence shows that Consumer obtained five different types of apples from Orchard, and Consumer later used two of those apples to bake a pie, and Consumer ate the pie, then there is substantial evidence that Consumer ate Orchard's apples -- even if it is not known *which* of Orchard's apples he ate.

124 Trial tr. at 2882 (Nov. 21, 2007). The Court further instructed the jury: "Thus, you must find in favor of TDY Industries on all of Mr. Tamraz's claims if you determine that Mr. Tamraz did not use any TDY products before December 12, 1992, or that any such use of TDY products did not lead to any harm to Mr. Tamraz before December 12, 1992. Further, during your deliberations, you are to consider the conduct of TDY Industries only before December 12, 1992." *Id.* at 2882-83.

125 In fact, in its post-judgment brief, TDY merely states that, after 1992, "another manufacturer" sold welding consumables under the McKay brand name; for

some reason, TDY is careful not to identify this manufacturer by name, possibly because it would tend to undermine Hobart's analogous post-judgment motion. Motion at 1 (docket no. 173). Today, Hobart sells McKay-branded welding rods. Solely for ease of reference, the Court refers in this opinion to the "other manufacturer" as Hobart, although this is immaterial to the Court's conclusion.

126 *Latex Glove*, 121 Cal.Rptr.2d at 309.

127 Trial tr. at 786 (Nov. 7, 2007). It is certainly a fair reading of this colloquy that Mr. Tamraz used McKay welding rods in 1992, which is when TDY manufactured them. Conceivably, Mr. Tamraz's last answer (A3) in this colloquy could be read as "separated" from the preceding question about the 1992 time-frame (Q1), but the jury had a basis to draw a reasonable inference that Mr. Tamraz's last answer continued to be responsive to the question about his welding experiences in 1992.

128 Mr. Tamraz testified on direct examination:

> Q. Look up at the screen. I think you have testified that you used the products of every one of those defendants whose logos are on the screen, correct?
>
> A. Yes, that's right. Teledyne McKay, is that the same one? The same product, right? Is that the same name?
>
> Q. Teledyne McKay?
>
> A. Yes. I have used every one.

Trial tr. at 829-30 (Nov. 7, 2006). While defendants assert this colloquy shows Mr. Tamraz was unsure whether the "McKay" welding consumables he used were actually manufactured by TDY's Teledyne McKay, it is just as reasonable to infer that Mr. Tamraz initially asked for clarification but then became sure of his identification, answering unequivocally. Defendants have also suggested informally that the Teledyne McKay logo shown to Mr. Tamraz was a mock-up created by counsel, and not a logo actually ever used by TDY. Even assuming this is true, it does not subtract from Mr. Tamraz's statement that he used a product with the Teledyne McKay name.

129 Mr. Tamraz testified on direct examination:

> A. For ENDI [Metal Works], I used P-5 or 5P6010.
>
> Q. Give us the name of the manufacturers, do you recall?
>
> A. The name of the manufacturer?
>
> Q. Yes.
>
> A. Lincoln, Hobart, Airco.
>
> Q. Any others?
>
> A. Linde, McKay. This is back earlier in my career, yes.

Trial tr. at 823 (Nov. 7, 2007).

130 Defendants also initially argued there was no such thing as a "McKay 30816 lime coat" welding consumable, which Mr. Tamraz testified he had used. *See* trial tr. at 857 (Nov. 7, 2007) ("I think I used some McKay 30816 lime coat for stainless steel welding."); *id.* at 2038 (Nov. 15, 2007) ("There is no such product as a McKay 30816 lime coat. I guess, you know, in all fairness, that could be -- there is a 308 product. There is a 316 product. But there is no such thing as a 30816."). Plaintiffs, however, produced a document showing TDY manufactured a welding rod with the AWS classification "E308-16." *See* docket no. 154, exh. C.

131 *Latex Glove*, 121 Cal.Rptr.2d at 309. (emphasis added).

132 Indeed, as plaintiffs point out, given that welding rods often sit in storage for a period of time after manufacture and purchase, it is quite possible that a "McKay" welding rod used in 1993 or later was manufactured by TDY.

133 Like defendants ESAB, Hobart, and TDY, BOC also argued that Mr. Tamraz did not offer sufficiently particularized evidence of which BOC welding consumables he used. Except to the extent stated below, for the same reasons this argument failed for ESAB, Hobart, and TDY, it fails for BOC.

134 Trial tr. at 2882 (Nov. 21, 2007). The Court further instructed the jury: "Thus, you must find in favor of the BOC Group, Inc. on all of Mr. Tamraz's claims if you determine that Mr. Tamraz did not use any Airco products before 1986. Further, during your

deliberations, you are to consider the conduct of the BOC Group only before 1986." *Id.*

135 Actually, Mr. Tamraz's testimony about products that he used "earlier in his career" can be read to refer to Airco consumables, as well. *See* footnote 129, above. Apparently, the jury so understood his testimony. The Court concludes, however, that this sole time-frame reference, unaccompanied by the sort of other testimony that Mr. Tamraz offered regarding TDY, does not provide the required quantum of substantial evidence.

136 ESAB motion at 1 (docket no. 171) (emphasis in original); TDY motion at 2 (docket no. 173) (emphasis in original).

137 Mr. Tamraz testified as follows:

> Q. Let's start with the warning labels, all right? You have seen a warning label prior to today, have you not?
>
> A. Yes.
>
> Q. And you first saw a warning label when you began welding. Is that true?
>
> A. Yes, real early.
>
> Q. And that would have been back in the '78, '79 time frame?
>
> A. '78, yeah.

> Q. So there is an issue about where they were placed, they should not have been placed here in the box or the can, but wherever they were placed, you saw a warning. Correct?
>
> A. Correct.

Trial tr. at 831 (Nov. 7, 2007) (cross examination).

138 Motion at 7-8 (docket no. 171) (citations omitted).

139 TDY's reply at 6 (docket no. 189).

140 Mr. Tamraz and his experts consistently criticized the defendants for placing their warnings in spots where welders would be less likely to see them, and for using typefaces that were difficult to read. *See. e.g.*, trial tr. at 492 (Nov. 5, 2007) (plaintiffs' counsel stating, during opening statement: "This is a warning label, Number 4, from Airco, one of the defendants. I will blow it up so it, you see, is easier to read, but this is part of the problem. This is real small print."); *id.* at 2262 (Nov. 16, 2007) (plaintiffs' counsel taking pains to show the jury that the warning was placed on the bottom of a 50-pound can). On these points, the Court instructed the jury: "In deciding whether directions, instructions or warnings were adequate, you should consider the[ir] location, language, and size," and "In assessing the adequacy of the warnings in this case, you are to consider the content of each warning given and also the mechanisms used to convey each such warning, such as its size and placement." Trial tr. at 2871, 2881 (Nov. 21, 2007).

141 *See Solis*, docket no. 194 (Draft Verdict Form at 1-3).

142 *See Solis*, docket no. 195 (Final Verdict Form at 1-2).

143 *See* case no. 03-CV-17000, master docket no. 1467 at 8 (Nov. 1, 2005) (Special Master's Ruling regarding the discoverability of the defendants' 1987 "Master Defense Agreement" and 2004 "Amended and Restated Master Defense Agreement," both of which contained "provisions . . . regarding apportionment of settlements and judgments [that] are fairly characterized as a 'judgment sharing agreement.'"). *See also Solis* trial tr. at 3238-39 and 3243-44 (defendants stating repeatedly they did not need compensatory damages broken out by defendant and that any liability would be joint and several; and plaintiffs' counsel suggesting this was because defendants had entered into a judgment sharing agreement); *id.* at 3245-46 (defense counsel confirming the Court's understanding that "the defendants are agreeing that if the jury comes back against the defendants collectively, that the defendants will collectively make sure that any judgment gets paid").

Following the *Solis* trial, this Court presided over the bellwether *Welding Fume* MDL trial of *Goforth v. Lincoln Electric Co.*, case nos. 06-CV-17217. Again, the defendants requested that the jury determine a *total* amount of compensatory damages, regardless of how many defendants the jury found liable, with any allocation of payment to be determined by defendants amongst themselves. *See Goforth* trial tr. at 4254-63 (Nov. 27, 2006); *Goforth* Verdict Form at 5 (dkt no. 135).

144 *See generally* trial tr. at 2672-76 (Nov. 20, 2007) (colloquy between the Court and defense counsel on this point). The colloquy ended as follows:

> THE COURT: * * * I want to make sure the record is clear, that this is the way you want the damages on the verdict form because really the defendants would have the right to ask for a much more detailed breakout, and given as I said that it was [defense counsel] last time that said you didn't want it, I don't want any issue in the Court of Appeals if you ever go there, that I should have broken that out and somehow you can't tell who the jury was punishing on the damages numbers. Do you understand?
>
> MR. HARBURG: Yes, Your Honor, I do, and for purposes of the damages - let me flip to it, to make sure I'm not missing something. Yes. We do agree that you can, that the jury can simply put in one number.
>
> THE COURT: For compensatory.
>
> MR. HARBURG: For compensatory. Just put in a bottom line number. The liability is broken out, and then if there is any allocation among the defendants, they will deal with that.

Id. at 2676.

145 Trial tr. at 2921-22 (Dec. 5, 2007).

146 *Id.* at 2922. As revealed by the colloquy set out below, the defendants reaffirmed and adopted this

approach again in *Jowers v. BOC Group, Inc.*, case no. 1:08-CV-36-KMO-JMR (S.D. Miss), which was the next MDL Bellwether trial after the *Tamraz* trial:

> THE COURT: I don't think they are necessary to the verdict form. I think the simpler the verdict form, the better. All those are elements that I have charged. The only thing I want to confirm about the verdict form, and this is important, because as I understand, [the Special Master] confirmed with the defendants, because the defendants did not propose in their verdict form that the dollar figures be, for anything other than punitive damages be apportioned out. We've gone through this before. It is important to make sure we're on the same page. The defendants have always requested that I not separately apportion damages for compensatories, and I want to make sure that's what the defendants want again this time.
>
> MR. ULMER: It is, Your Honor.
>
> THE COURT: Okay. All right. Great. On that issue, because it is late, I'm not going to go into it, but I want to incorporate all the discussions I have had with the defendants about the implications of that decision from the prior cases. And Mr. Harburg is shaking his head yes. Okay. See you tomorrow.

Jowers trial tr. at 3124-25 (Mar. 2, 2008).

Nos. 08-4015/4016

UNITED STATES COURT OF APPEALS
FOR THE SIXTH CIRCUIT

JEFF TAMRAZ, ET AL., Plaintiffs-Appellees,
v.
LINCOLN ELECTRIC COMPANY, ET AL.,
Defendants-Appellants.

November 9, 2010, Filed

BEFORE: MARTIN and SUTTON, Circuit Judges; and REEVES, * District Judge.

ORDER

The court having received a petition for rehearing en banc, which was circulated to all active judges of this court, none of whom requested a vote on the suggestion for rehearing en banc, the petition for rehearing has been referred to the original panel.

The panel has further reviewed the petition for rehearing and concludes that the issues raised in the petition were fully considered upon the original submission and decision of the cases. Accordingly, the petition is denied. Judge Martin would grant rehearing for the reasons stated in his dissent.

Footnote

*Hon. Danny C. Reeves, United States District Judge for the Eastern District of Kentucky, sitting by designation.

Case 1:04CV18948
MDL Docket No. 1535

UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF OHIO
EASTERN DIVISION

JEFFREY A. TAMRAZ, et al. Plaintiffs,

VS.

LINCOLN ELECTRIC COMPANY, et al. Defendants.

VOLUME 1

PAGES 1 to 184
Filed: 04/30/2009

TRANSCRIPT OF PROCEEDINGS HAD BEFORE THE HONORABLE JUDGE KATHLEEN M. O'MALLEY, JUDGE OF SAID COURT, ON THURSDAY, NOVEMBER 1ST, 2007, COMMENCING AT 1:00 O'CLOCK P.M.

Court Reporter:

JUDITH A. GAGE, RMR-CRR
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SUITE 7-189
CLEVELAND, OHIO 44113, (216) 357-7238

PROCEEDINGS

THE COURT: Good afternoon. We're having technical difficulties already, and the trial has not even started.

We're here for a final pretrial in this matter. Let me ask you how to pronounce the plaintiff's name. Everybody is saying Tamraz, but that is not how it is spelled.

MR. CLIMACO: Your Honor, I am one of those. I have been corrected by my co-counsel. It is Tamraz. That doesn't mean that a few times I won't say Tamraz.

THE COURT: I just realized that David and I have been for the last several days using the name, and then I looked at the spelling again this morning and realized we were not pronouncing it properly or appeared to be.

Okay. We have a number of issues to address. There are a number of motions in limine. We'll go through again some general pretrial matters. Most of you are very familiar with them. And we'll talk briefly about the summary judgment motions

As with Goforth, I see very little reason for me to have argument on most of these issues. I might have a few questions relating to some of them, the ones that are new. But as to the bulk of these motions, they either raise issues that we have seen in the past, even though sometimes there are some new arguments presented, or they raise issues with which even though not framed in the exact same way but with which I have sufficient familiarity that I think I can rule without much discussion.

I'm going to start with the motions, the pretrial motions. Pretty much we will go according to docket numbers with one exception, and that is I'm going to skip the first one, which is Docket Number -- I'm sorry, that's the second one. All right.

Let's start with Docket Number 55, which is the first of the pretrial motions, it is defendants' motion for order to exclude the plaintiffs' experts. This is simply a reiteration of arguments the defendants have made in the past with respect to a number of the plaintiffs' experts and specifically Cunitz, Longo, Parent and Rosen.

I have already addressed all the arguments presented by the defendants and the defendants' current briefing candidly concedes that. The defendants ask me to adopt all portions of my prior rulings that exclude any portions of that testimony, and to rethink those portions of my prior rulings that allow any portions of the testimony by those individuals. Plaintiffs simply ask me to stand by my earlier rulings. The motion is denied.

I will stand by all of the earlier rulings that I have made with respect to those expert witnesses. As I said, they are Cunitz, Longo, Parent and Rosen, all the defendants' earlier objections to their testimony are preserved, and all the restrictions that the Court placed on the testimony of those experts will remain intact for purposes of this proceeding, and you can refer to the Court's earlier rulings, some written and some oral, relating to those experts, but I think we all know what the limitations are.

Okay. Next is Docket Number 56, defendants' motion for order excluding portions of Dr. Carlini's opinions. The briefing on this was certainly extremely well written from both sides, and the parties raise a number of issues, both factual and legal in them. I have read all of the briefs. I have read all the depositions, or the two depositions that were take of Dr. Carlini.

I have gone back and reread the Court's Daubert opinion, which was on the main MDL docket at 1353, and I have decided that I am going to deny the defendants' motion.

To a large extent, the defendants' motion asks the Court to draw bright lines regarding diagnoses of movement disorders that I have already declined to (?) draw, and I have already decided that the current state of the science does not require to be drawn.

I find that Dr. Carlini is a well-qualified physician, extremely well educated, with an established practice. I see nothing lacking in his qualifications to render opinions regarding movement disorders, and I see nothing about his methodology which is either flawed or inconsistent with the very diagnostic methods that other experts in this case, both the plaintiffs and the defendants' experts alike, have used and have described as appropriate diagnostic methods.

I think it is telling that the defendants have not proffered an affidavit from any expert indicating that there is anything wrong with Dr. Carlini's methodology, or that there is anything faulty with respect to his qualifications.

The fact that the defendants may disagree with Dr. Carlini's opinions or his word choice or the fact that his word choice may not be as cautious as one might expect from a practiced litigation expert to me is insufficient grounds to exclude his testimony.

It is clear that the defendants have fair grounds to attack the somewhat unusual. diagnosis that Dr. Carlini

renders in this case, and that they have done so in their cross-examination of Dr. Carlini, but that to me goes to the weight and not to the admissibility of his testimony. So I am not going to exclude either portions of Dr. Carlini's testimony or, as was hinted in the plaintiffs' response, the entirety of Dr. Carlini's testimony. I'm going to allow his testimony and deny the defendants' motion.

Next is Docket Number 62, defendants' motion in limine to exclude evidence regarding defendants' opposition to defendants' motion for a Court ordered epi study. That is granted as unopposed. It was granted previously in Solis and Goforth. The plaintiffs don't oppose the Court entering the same order on in this case.

Next is docket 63, defendants motion in limine to exclude animations in plaintiffs' opening argument. In both the Solis case and Go forth case, this issue came up at pretrial, and the Court sustained the defendants' motion in part, restricting use of a particular animation. I assume that's the only animation the defendants are objecting to here. Is that correct? I don't know who I'm supposed to turn to.

Mr. Kennedy?

MR. KENNEDY: Yes.

THE COURT: The Court barred the plaintiffs

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No. 10-1122

SUPREME COURT, U.S.
FILED
APR 18 2011

In The Supreme Court of the United States

JEFF TAMRAZ AND TERRY TAMRAZ,
Petitioners,

v.

LINCOLN ELECTRIC COMPANY; HOBART BROTHERS COMPANY; ESAB GROUP, INC. AND TDY INDUSTRIES, INC.,
Respondents.

On Petition For A Writ Of Certiorari To The United States Court Of Appeals For The Sixth Circuit

BRIEF IN OPPOSITION

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Questions Presented

1. Whether the court of appeals properly ruled that a doctor's opinion that manganese exposure caused the plaintiff to develop Parkinson's disease – a disease with no known cause – was unreliable and therefore inadmissible because it was, in the doctor's own words, "speculative."

2. Whether the court of appeals applied the right standard in ruling that the district court abused its discretion by admitting unreliable expert testimony.

Corporate Disclosure Statement

Pursuant to Supreme Court Rule 29.6, respondents state that The Lincoln Electric Company is a wholly-owned subsidiary of Lincoln Electric Holdings, Inc., a publicly traded company. No publicly traded company owns more than 10% of the stock of Lincoln Electric Holdings, Inc. Hobart Brothers Company is a wholly-owned subsidiary of Illinois Tool Works Inc., a publicly traded corporation. No publicly traded company owns more than 10% of the stock of Illinois Tool Works Inc. The ultimate parent company of The ESAB Group, Inc. is Charter International plc, a publicly held company traded on the London Stock Exchange. No publicly held company owns more than 10% of the stock of Charter plc. TDY Industries, Inc., is a wholly owned subsidiary of TDY Holdings LLC, which is a wholly owned subsidiary of Allegheny Technologies Incorporated. No publicly traded company owns more than 10% of the stock of Allegheny Technologies Incorporated.

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INTRODUCTION AND STATEMENT OF THE CASE

The court of appeals' holding in this case – that the trial court abused its discretion in admitting medical causation testimony from a physician who described his own opinions as "speculative" – was neither remarkable nor controversial. Rather, it derived directly from this Court's ruling in *Daubert v. Merrell Dow Pharmaceuticals, Inc.* that scientific "'knowledge' connotes more than subjective belief or unsupported speculation." 509 U.S. 579, 590 (1993). Nonetheless, petitioners conjure up a supposed circuit split on the admissibility of a doctor's "differential etiology" and argue that the court of appeals covertly applied a *de novo* standard of review instead of the required abuse-of-discretion standard.

Neither issue warrants this Court's review. Petitioners have not identified any disagreement among the lower courts or departure from this Court's precedents. Although they assert – with almost no explanation – that the ruling below applied a higher standard for differential etiologies than is required by the Third or Fourth Circuits, that argument misconstrues the ruling below and ignores harmonious decisions from both circuits. Nor is there any merit to petitioners' allegation that the court of appeals departed from this Court's precedents by applying a *de novo* standard of review. Indeed, the court of appeals expressly stated that it was applying an abuse-of-discretion standard. Petitioners call this "lip service" (Pet. 15), echoing the dissent below. But the dissent advocated a level of deference to district courts that has been expressly rejected by courts of appeal and by Justices of this Court.

In short, speculative testimony has no place in the courtroom – and the court of appeals properly found that the district court abused its discretion in admitting medical causation testimony that was premised on multiple "speculative jumps."

1. The Welding Process. Respondents The Lincoln Electric Company, Hobart Brothers Company, The ESAB Group, Inc. and TDY Industries, Inc. are current and former manufacturers of welding consumables. Welding is the process of joining two pieces of "base metal" by heating their surfaces while adding new molten metal. The vast majority of industrial welding has always involved mild steel, which is composed of iron plus a small amount of carbon and other alloys. One such alloy is manganese, which gives steel toughness and hardness, and de-oxidizes and de-sulfurizes the steel. To obtain an effective weld, the welding material or "consumable" used to join the two pieces of base metal must be compatible with the base metal. Because most steel contains manganese, manganese has been present in most welding consumable products from the inception of welding, and it has no substitute.

The high heat generated in the welding process necessarily releases particulate fumes, known as "welding fumes." Because manganese is present in both the base metal and the welding consumables, manganese is one of the elements in welding fumes.

Nobody disputes that exposure to certain levels of manganese is safe. In fact, manganese is an essential nutrient. At extremely high levels of exposure, however, manganese can become toxic and can cause, in certain cases, a very rare neurological disease called manganism. Manganism is distinct from an-

other neurological condition, Parkinson's disease ("PD") – an "idiopathic" disease that has no known cause or explanation and afflicts more than a million Americans.

2. Welding Fume Litigation. About a decade ago, a group of plaintiffs' lawyers, working with a neurologist, Dr. Paul Nausieda, began conducting mass medical screenings of welders and filing lawsuits, alleging that exposure to manganese in welding fumes had caused the welders to develop a rare neurological disease known as manganism. With the number of welding fume cases rising, the Judicial Panel on Multidistrict Litigation created a multidistrict litigation proceeding before Judge Kathleen O'Malley of the United States District Court for the Northern District of Ohio to coordinate welding fume cases for pretrial proceedings. *In re Welding Rod Prods. Liab. Litig.*, 269 F. Supp. 2d 1365 (J.P.M.L. 2003).

3. Petitioners' Suit. In September 2004, petitioners sued several current and former welding consumable manufacturers, contending that as a result of exposure to manganese and manganese compounds in welding fumes, Mr. Tamraz developed permanent and severe neurological damage. Petitioners alleged that the labels on the products failed to warn of this danger. Their case was consolidated with ongoing multidistrict litigation and was selected as one of several "bellwether trials" that would guide the resolution of the other cases.

At trial, petitioners called Dr. Nausieda, who testified that the plaintiff has manganism. They also introduced the testimony of Dr. Walter Carlini, Mr. Tamraz's treating physician. Dr. Carlini diagnosed Mr. Tamraz with PD – a valid and reliable diagnosis

that respondents did not challenge. But he did not stop there. Instead, Dr. Carlini also offered a hypothesis as to the *cause* of Mr. Tamraz's disease: that he had an unusual genetic predisposition to PD that was triggered by exposure to manganese in welding fumes. There was no science to support this hypothesis; as Dr. Carlini himself admitted, it was based on sheer speculation. Dr. Carlini simply professed faith that some day in the future, researchers will identify a link between genetics, manganese exposure and PD.

Respondents moved to exclude Dr. Carlini's causal opinion, citing his admission that it was based solely on his own speculation, rather than any published literature or scientific studies. Petitioners responded that Dr. Carlini had offered a reliable opinion based on a differential diagnosis. Their primary argument was that Dr. Carlini had actually diagnosed Tamraz with "manganese-induced parkinsonism," which, they argued, was simply another term for manganism. Since manganism is caused only by manganese exposure, they posited, a reliable diagnosis of the illness obviated the need for a reliable analysis of its cause. But this argument was not based in reality, because Dr. Carlini was clear that he thought Mr. Tamraz had PD – not manganism. The term manganese-induced parkinsonism was simply a label he attached to Mr. Tamraz's PD to emphasize his belief that it was caused by manganese exposure.

Petitioners also argued in the alternative that, assuming Dr. Carlini had diagnosed PD rather than manganism, his causation opinion could be supported by scientific literature. But it was literature with which Dr. Carlini was entirely unfamiliar. As far as

Dr. Carlini knew, science did not accept any connection between manganese exposure and PD, a disease with no cause known to science. And Dr. Carlini had not done any of his own research to establish such a link. Nonetheless, the trial court summarily denied respondents' motion without oral argument and without any written order. (Pet. App. D, at 131a-132a.)

Throughout the trial, petitioners relied heavily on Dr. Carlini's admittedly speculative causation opinion to argue that respondents' products were the proximate cause of Mr. Tamraz's injuries. Indeed, Dr. Carlini's untested hypothesis became the centerpiece of petitioners' case. During closing arguments, for example, petitioners' counsel told the jury that "Dr. Carlini alone proves by a preponderance of the evidence that this man has manganese-induced parkinsonism." (*Id.* at 22a.)

The jury ultimately returned a verdict in favor of petitioners, awarding $20.5 million in damages. (*See* Pet. App. B, at 44a.) The defendant manufacturers filed motions to overturn this verdict under Rule 50 of the Federal Rules of Civil Procedure, but the district court denied the challenges of every defendant except The BOC Group, Inc., against whom the court found insufficient evidence to sustain the verdict. (*Id.*)

4. Appellate Proceedings. Respondents appealed, arguing, *inter alia*, that the district court should not have admitted Dr. Carlini's opinion that their products caused Mr. Tamraz's PD because the testimony did not satisfy the requirements of Rule 702 of the Federal Rules of Evidence. (Pet. App. A, at 4a.) The Sixth Circuit agreed, holding that admission of Dr.

Carlini's causation opinion was an "abuse of discretion" because it involved multiple "speculative jumps." (See *id.* at 4a, 10a, 13a (noting that "Dr. Carlini's opinion contains not just one speculation but a string of them").)

The court of appeals spelled out each of the four speculative links in Dr. Carlini's theory of causation. First, the court found, Dr. Carlini merely hypothesized that "toxins combined with genetics may cause [some] cases of PD." (*Id.* at 9a.) Indeed, he "described the literature hypothesizing a link between environmental toxins and latent genetic Parkinson's Disease as 'all theoretical.'" (*Id.* at 10a.) Next, Dr. Carlini theorized that manganese is "a possible candidate for triggering" PD because it "is known to cause manganism" (*id.* at 9a), although "he conceded he knew of no studies finding a link between manganese and Parkinson's Disease and that 'studies that have looked at that . . . have not found a very strong correlation.'" (*Id.* at 10a.) Then, Dr. Carlini posited that Mr. Tamraz "may have the genes for" PD in a leap that he himself conceded was "speculation." (*Id.* at 9a-10a.) And finally, he guessed that manganese "may have triggered these genes" and caused Mr. Tamraz's PD. (*Id.* at 9a.)

The court of appeals found this last step particularly problematic. It noted that "Dr. Carlini never explained how he made this leap – how *this* case stemmed from manganese exposure." (*Id.* at 11a.) This was problematic because, even if "manganese *could cause* Parkinson's Disease in someone like Tamraz[, that] does not show that manganese *did cause* Tamraz's" PD. (*Id.* at 10a-11a.) Indeed, given the science, the presumption would have to be the

opposite – "Parkinson's Disease occurs commonly in the general population and usually without any known cause. Any given case of Parkinson's Disease thus might have occurred regardless of manganese exposure, making it hard to attribute one case to manganese over all of the other possible causes." (*Id.* at 11a.) The "closest" thing to an explanation offered by Dr. Carlini was the "early onset" of PD in Mr. Tamraz. (*Id.* at 11a-12a.) But the court of appeals rightly rejected this analysis as insufficiently rigorous, observing that Dr. Carlini himself acknowledged that ten percent of PD cases develop before age 50, "which, considering the high prevalence of Parkinson's Disease, does not create an inference that something particularly unusual must have caused it in Tamraz, who was 41 to 44 years old at the onset of his symptoms." (*Id.* at 12a.)

For all of these reasons, the Sixth Circuit concluded that Dr. Carlini's causal opinion suffered "from a lack a foundation both for why manganese could cause Parkinson's Disease and why manganese caused this case of Parkinson's Disease." (*Id.* at 12a.) The court carefully acknowledged that Rule 702 "does not require anything approaching absolute certainty." (*Id.* at 13a.) "Yet, so long as there is a line, some forms of testimony may cross it, and that happened here." (*Id.*) The court again emphasized the fact that Dr. Carlini's testimony was admittedly "speculative" in multiple respects, resulting in significant gaps in his reasoning. (*Id.*) And such reasoning – where "A suggests by analogy the possibility of B, which might also apply to C, which, if we speculate about D, could eventually trigger E" – is inadmissible under Rule 702. (*Id.*) "At some point, the train becomes too long to pull and the couplings

too weak to hold the car together." (*Id.*) Accordingly, the trial court erred in admitting the testimony.

Finally, in light of the emphasis that plaintiffs placed upon Dr. Carlini's testimony at trial, the court of appeals further determined that this error substantially affected the judgment in favor of the plaintiffs and remanded for a new trial. (*Id.* at 22a-24a ("Given the importance of Dr. Carlini's testimony, we cannot say with 'fair assurance' that the result would have been the same without it.").)[1]

Judge Martin dissented, expressing his belief that the majority had not afforded sufficient deference to the district court. (*Id.* at 27a.) But the dissent appeared to embrace something more akin to abdication than deference. It would have held that a court of appeals has no authority to reverse a *Daubert* ruling admitting expert evidence unless "it is absolutely clear that the testimony is nothing more than 'junk science' that the jury cannot be trusted to evaluate." (*Id.* at 32a.) In practice, this would be no role at all since, in the dissent's view, "[i]t is for the district court to determine whether expert testimony is essentially 'junk science.'" (*Id.* at 35a.) In other words, a district court's decision to admit expert evidence would be all but unreviewable. Apparently, no other judges on the court were willing to accept this stance; petitioners requested that the Sixth Circuit rehear the case en banc, but only Judge Martin would have granted rehearing. (Pet. App. C, at 127a.) Petitioners now seek this Court's discretionary review.

---

[1] Petitioners do not dispute this portion of the Sixth Circuit's ruling.

REASONS FOR DENYING THE PETITION

The ruling of the court of appeals is in harmony with those of other circuits and this Court's precedents. It was also rightly decided – under the proper standard – leaving nothing for this Court to correct and no compelling argument for it to grant review.

I. The Sixth Circuit's Ruling Does Not Conflict With Those Of Other Circuits Regarding The "Ruling Out" Phase Of Differential Etiologies.

Petitioners first contend that this Court's review is needed to resolve a purported split among the courts of appeals that resulted from the ruling below. Not so. No split exists, and petitioners' argument to the contrary rests on a tortured reading of the decision of the court of appeals.

According to petitioners, Dr. Carlini's opinion was the result of a reliable differential etiology – that is, a methodical analysis in which all possible causes were ruled in and then ruled out one by one until the remaining cause is left as the most likely. Petitioners contend that other courts of appeals that have considered this methodology have properly held that causation experts need not definitively "'rule out every possible cause of the plaintiff's illness'" (Pet. 28 (quoting *Heller v. Shaw Indus., Inc.*, 167 F.3d 146, 156 (3d Cir. 1999))), while the court of appeals in this case "requires them to rule out with conclusive finality all other unknown agents or to prove every inference or assumption in their thinking" (*id.* 27-28).

But the ruling below holds nothing of the sort. Indeed, it expressly disclaimed a requirement of conclusive finality, acknowledging that Rule 702 "does

not require anything approaching absolute certainty." (Pet. App. A, at 13a.) Nor did it adopt a general approach to differential etiologies that requires an expert to affirmatively rule out all causes but one. To the contrary, it cited with approval prior Sixth Circuit decisions holding that differential etiologies were admissible despite alleged infirmities, including one doctor's opinion that had only "ruled out *most* alternative causes" because the factors he had not ruled out were not shown to be likely causes of the injury at issue. (*Id.* at 20a (emphasis added) (citing *Best v. Lowe's Home Ctrs., Inc.*, 563 F.3d 171, 179 (6th Cir. 2009)).) *See also Best*, 563 F.3d at 179 ("the core of differential diagnosis" is only "a requirement that experts at least *consider* alternative causes") (emphasis added) (quoting *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 759 (3d Cir. 1994)).

Here, the problem for petitioners under the facts of their specific case was that "unknown (idiopathic) causation currently accounts for the vast majority of Parkinson's Disease cases, making it impossible to ignore and difficult to rule out." (Pet. App. A, at 20a (punctuation omitted).) Petitioners argue that Mr. Tamraz's early onset and his occupational history support Dr. Carlini's conclusion that Mr. Tamraz's PD was more likely caused by manganese exposure than idiopathic in origin. (Pet. 26-28.) But early onset occurs in "roughly ten percent of people" who develop PD; thus, "considering the high prevalence" of PD, early onset "does not create an inference that something particularly unusual must have caused it in Tamraz." (Pet. App. A, at 12a.) Petitioners also contend that respondents' experts agree that early onset is a sign of a genetic predisposition (Pet. 26); but that is only with respect to developing PD gener-

ally, not developing PD *as a result of exposure to manganese*. As the court of appeals pointed out, Dr. Carlini admitted that "the base probability of getting parkinsonism from" a genetic predisposition "is unknown." (Pet. App. A, at 11a.)

Nor does Mr. Tamraz's occupational history change the analysis. Because Dr. Carlini knew of no study that establishes any correlation between manganese exposure and PD, he had no basis to conclude (or even guess) that exposure to manganese would meaningfully increase the chances of developing PD. Thus, Dr. Carlini's supposed bases for ruling out an idiopathic origin as the most likely cause of Mr. Tamraz's condition lacked any scientific support. *See Bland v. Verizon Wireless, (VAW) LLC*, 538 F.3d 893, 897 (8th Cir. 2008) ("Where the cause of the condition is unknown in the majority of cases, [the expert] cannot properly conclude, based upon a differential diagnosis, [that the plaintiff's] exposure to freon was 'the most probable cause' of [his] exercise-induced asthma."). (*See also* Pet. App. A, at 11a (citing *Bland* and another similar case with approval).)

Contrary to petitioners' suggestion, the Sixth Circuit's approach was not in conflict with that of the Third and Fourth Circuits. All three courts require that a differential etiology or diagnosis satisfy a minimum "reliability" threshold. Compare Pet. App. A, at 18a, with *Heller*, 167 F.3d at 157 (noting that "some reliable basis for a causation conclusion must exist"), and *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 265-66 (4th Cir. 1999) (citing precedent for the proposition that, where a defendant points to an alternative plausible cause other than the defendant's actions, it "becomes necessary for the

plaintiff's expert to offer a good explanation as to why his or her conclusion remains reliable") (internal quotation marks and citation omitted). And like the Sixth Circuit, the Third and Fourth Circuits have rejected differential etiologies and diagnoses that they determined did not meet this minimum threshold. *See, e.g.*, *Cooper v. Smith & Nephew, Inc.*, 259 F.3d 194, 202 (4th Cir. 2001) (affirming exclusion of unreliable causation opinion purportedly based on differential etiology because, among other things, it failed to rule out alternative causes, and a differential etiology is not reliable where "an expert utterly fails to consider alternative causes or fails to offer an explanation for why the proffered alternative cause was not the sole cause"); *Feit v. Great W. Life & Annuity Ins. Co.*, 271 F. App'x 246, 254 (3d Cir. 2008) (holding that expert's theory of cause of death was not based on a reliable differential diagnosis because the expert failed to consider and rule out all possible causes). In short, each of these jurisdictions applies identical requirements to differential etiologies.[2] That is reason enough to deny the petition.

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[2] So do the other courts of appeals. *See, e.g.*, *Guinn v. Astrazeneca Pharms. LP*, 602 F.3d 1245, 1253 (11th Cir. 2010) (indicating that, in order for a differential diagnosis to be admissible, it needs to provide a "reliable" basis for an opinion on causation); *Ervin v. Johnson & Johnson, Inc.*, 492 F.3d 901, 904 (7th Cir. 2007) (upholding district court's determination that an expert's differential diagnosis "was tainted by critical flaws leaving his opinions unreliable under the standards set forth in Rule 702 and *Daubert*") (internal quotation marks and citation omitted); *Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 886 (10th Cir. 2005) ("Plaintiff's experts' differential diagnoses and case studies are scientifically unreliable because they as-

There is another reason as well. Even if petitioners had demonstrated a genuine split, this case would not be a proper vehicle to resolve it. After all, petitioners contend that the decision below created a split over whether a doctor performing a differential etiology must rule out "all possible alternative causes" with "conclusive finality." (Pet. 28.) But the court of appeals determined that Dr. Carlini's opinion was unreliable as to *both* the rule-in and rule-out phases of his analysis. (Pet. App. A, at 18a-19a ("Dr. Carlini's opinion fails the last two prongs because . . . his efforts to 'rule in' manganese exposure as a possible cause or to 'rule out' other possible causes turned on speculation, not a valid methodology.").) No division in authority is claimed as to the "rule-in" standard applied by the court of appeals, and thus no basis is presented for its review. *See, e.g.*, Eugene Gressman et al., *Supreme Court Practice* § 4.4(f), at 248 (9th ed. 2007) ("If the resolution of a clear conflict is irrelevant to the ultimate outcome of the case before the Court, certiorari may be denied.") (citing

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sume . . . a causal connection[.]"); *Amorgianos v. Nat'l R.R. Passenger Corp.*, 303 F.3d 256, 265 (2d Cir. 2002) (holding that, where an expert opinion is based on data or studies that are inadequate to support the conclusions reached, *Daubert* and Rule 702 mandate exclusion of that "unreliable" opinion testimony); *Glastetter v. Novartis Pharms. Corp.*, 252 F.3d 986, 991 (8th Cir. 2001) (noting the difficulty in "reliably extrapolating" from the results of a study to prove causation in a differential diagnosis); *Moore v. Ashland Chem., Inc.*, 151 F.3d 269, 275 (5th Cir. 1998) (en banc) (indicating that, where a proposed expert conducts a differential diagnosis, the causation opinion must be based on "good grounds," which equates to a standard of "evidentiary reliability").

Sommerville v. United States, 376 U.S. 909 (1964)). For this reason too, certiorari should be denied.

II. The Sixth Circuit's Ruling Does Not Depart From This Court's Precedent Regarding The Standard Of Review.

Petitioners also contend that review is needed because the court of appeals departed from this Court's precedent establishing an abuse-of-discretion standard for appellate review of district court rulings under Federal Rule of Evidence 702. (Pet. 29-31.) In so arguing, petitioners ignore the court's explicit invocation of the abuse-of-discretion standard (*see* Pet. App. A, at 4a ("we review . . . for abuse of discretion")), and instead echo the dissent's accusation that the majority in fact applied *de novo* review (*see* Pet. App. A, at 30a; Pet. 15).

But it was the dissent (and now petitioners) that advocated an improper standard of review – not the majority. Under the dissent's approach, courts of appeals would be barred from reversing a decision to admit expert testimony unless it is "absolutely clear that the testimony is nothing more than 'junk science'"; at the same time, the dissent would commit the exclusive authority to decide what is "junk science" to the district courts. (Pet. App. A, at 32a, 35a.) That leaves no role for the courts of appeals at all. As long as a district court does not affirmatively declare evidence to be "junk science" and then proceed to admit it at trial regardless, its decision would be unreviewable.

That is not the law. In *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152 (1999), the Court reaffirmed "the importance of *Daubert*'s gatekeeping require-

ment," explaining that a district court has a duty "to ensure the reliability and relevancy of expert testimony." Three Justices concurred fully in the judgment but wrote separately to emphasize this point, noting that the district court's discretion in performing its role "is not discretion to abandon the gatekeeping function." *Id.* at 158-59 (Scalia, J., concurring); *see also id.* at 159 ("I think it worth adding that it is not discretion to perform the function inadequately."). The courts of appeals share this understanding – that the abuse-of-discretion standard does not mean insulation from review – and have reversed district courts for admitting unreliable expert testimony where appropriate. *See, e.g.*, *Cruz-Vazquez v. Mennonite Gen. Hosp., Inc.*, 613 F.3d 54, 57 (1st Cir. 2010) (noting that a trial court's "discretion is not without bounds"); *McClain v. Metabolife Int'l, Inc.*, 401 F.3d 1233, 1238-55 (11th Cir. 2005) (reversing and remanding for a new trial where the district court abused its discretion by allowing plaintiffs' experts in a toxic tort case to take "leaps of faith"); *Joy v. Bell Helicopter Textron, Inc.*, 999 F.2d 549, 567-69 (D.C. Cir. 1993) (reversing district court's admission of expert testimony in wrongful-death case where the testimony was highly speculative); *In re Air Crash Disaster at New Orleans, La.*, 795 F.2d 1230, 1233-35 (5th Cir. 1986) (same); *Gumbs v. Int'l Harvester, Inc.*, 718 F.2d 88, 98-99 (3d Cir. 1983) (reversing and remanding for a new trial where, *inter alia*, the district court admitted expert testimony that lacked a sufficient factual foundation). The Sixth Circuit did the same here – and did so under the proper standard.

Although petitioners do not identify any particular aspect of the court of appeals ruling that

supposedly reflects *de novo* review, they do contend elsewhere in their petition that the court of appeals made three errors (in addition to the alleged error in its "rule-out" analysis, discussed in Part I, *supra*). To the extent petitioners intend to argue that these three supposed errors reflect application of an improper standard, their arguments fail – badly.

First, petitioners defend Dr. Carlini's reliance on literature speculating about a link between environmental toxins such as manganese and a genetic predisposition to PD. (*Id.* 23.) But Dr. Carlini himself admitted that this was "all hypothetical" rather than proven science. (Pet. App. A, at 10a.) That concession alone justifies the Sixth Circuit's conclusion. *Cf. J.B. Hunt Transp., Inc., v. Gen. Motors Corp.*, 243 F.3d 441, 444 (8th Cir. 2001) ("Because of the deficiencies at the core of his opinion, including his own admission concerning his inability to scientifically reconstruct the accident, the [expert's] resulting conclusion . . . was mere speculation and pure conjecture."). Petitioners counter that other scientists believe PD is "most likely caused by a combination of genetic and environmental factors," citing an article by Dr. Irene Litvan and respondents' expert, Dr. Anthony Lang, among others. (*See* Pet. 23-24 (citing Litvan et al., *The etiopathogenesis of Parkinson's disease and suggestions for future research: Part I*, J. Neuropathol. Exp. Neurol. 66(4):251-257 (2007)).) But the second part of the same article refers to this belief as a "hypothesis." Litvan et al., *The etiopathogenesis of Parkinson's disease and suggestions for future research: Part II*, J. Neuropathol. Exp. Neurol. 66(5):329-336 (2007). The article concedes, moreover, that "despite a large number of studies" of "candidate genes," the "great

majority of the published associations in the area of PD have not been replicated." *Id.* Indeed, petitioners' own word choice betrays the speculative nature of this evidence, claiming that the genetics hypothesis is an "evolving" one that is "emerging" in "a lot of studies" and in "a lot of thinking." (*See* Pet. 23.) Thus, the court of appeals rightly concluded that this part of Dr. Carlini's opinion was "no more than a hypothesis" that did not qualify as "'knowledge.'" (Pet. App. A, at 9a.)

Petitioners next defend Dr. Carlini's hypothesis that, because manganese is known to cause manganism, it would be a possible candidate for triggering PD as well. (Pet. 24.) But Dr. Carlini conceded that he knew of no studies that found a link between manganese and PD. (Pet. App. A, at 10a.) Petitioners complain that the particular concession quoted by the Sixth Circuit came from testimony about studies looking at manganism rather than PD. (Pet. 24.) But this is a red herring – the Sixth Circuit cited numerous other parts of Dr. Carlini's testimony that it did not quote, and these portions made clear that he could not cite "a single study" to support his belief that manganese could combine with genetic factors to cause or accelerate PD because such studies "don't exist yet." (J.A. 599.) Moreover, petitioners do not dispute the Sixth Circuit's observation that the studies that have looked for a link between manganese and PD have found nothing. (Pet. App. A, at 10a ("'Epidemiological studies have failed to find a correlation between manganese and Parkinson's disease.'") (quoting J.A. 623).) As they did below, petitioners fail to identify even one countervailing study establishing a link between manganese and PD. (*See* Pet. 24.)

Finally, petitioners defend Dr. Carlini's hunch that Mr. Tamraz may have had the hypothesized genetic predisposition to PD. (*Id.* 24-25.) Dr. Carlini, however, openly conceded that it was only "speculation" – not hard scientific proof – that led him to guess that Mr. Tamraz had an "underlying predisposition" to PD, even though Mr. Tamraz has no family history of PD. (Pet. App. A, at 10a.) Notably, Dr. Carlini performed no genetic tests, did not know what to test for, and did not know how much a hypothesized predisposition would increase someone's odds of getting PD. (*Id.* at 11a (noting that Dr. Carlini only explained "tests he *might* do, not tests he had done").) Petitioners counter that because "there are *probably*" a number of people who have a genetic predisposition to PD but never get the disease, there *could have been* members of Mr. Tamraz's family who were predisposed to develop PD and Mr. Tamraz could have inherited a genetic predisposition from them. (*See* Pet. 24-25 (emphasis added).) This argument, in and of itself, is "speculative three times over." (Pet. App. A, at 10a (citing *Siharath v. Sandoz Pharm. Corp.*, 131 F. Supp. 2d 1347, 1371 (N.D. Ga. 2001) ("Three scientifically unwarranted 'leaps of faith' exist in this causal chain."), *aff'd*, 295 F.3d 1194 (11th Cir. 2002)).) Indeed, the most petitioners can say with respect to this tenuous reasoning is that it does not "conclusively defeat [Dr. Carlini's] overall theory of causation." (Pet. 25.)

In short, "the district court enjoys broad discretion over where to draw the line" on the admissibility of expert testimony, but "so long as there is a line, some forms of testimony may cross it, and that happened here." (Pet. App. A, at 13a.) Dr. Carlini's causation testimony was nothing more than "guess-

work," and "'the courtroom is not the place for scientific guesswork, even of the inspired sort.'" (Pet. App. A, at 12a-13a (quoting *Rosen v. Ciba-Geigy Corp.*, 78 F.3d 316, 319 (7th Cir. 1996)).) For these reasons, the district court *did* abuse its discretion in admitting Dr. Carlini's causation opinion, the court of appeals reversed and remanded under the proper standard, and there is no reason for this Court to disturb that judgment.

CONCLUSION

For the foregoing reasons, the Court should deny the petition for a writ of certiorari.

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No. 10-1122

In the Supreme Court of the United States

JEFF TAMRAZ AND TERRY TAMRAZ,
Petitioners,

v.

LINCOLN ELECTRIC COMPANY; HOBART BROTHERS COMPANY; ESAB GROUP, INC. AND TDY INDUSTRIES, INC.,
Respondents.

On Petition for a Writ of Certiorari to the United States Court of Appeals for the Sixth Circuit

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In their Brief in Opposition, respondents insist that the majority in this case understood and applied the "gatekeeper" function and the abuse-of-discretion standard of review precisely as this Court's opinions in *Daubert*,[1] *Kumho Tire*,[2] and *Joiner*[3] intended. Not only is the majority's opinion a stark departure from the analytical regime established by those opinions, it also cannot be reconciled with the way other Circuits apply the very same regime. The latest illustration of this irreconcilable conflict is the recent decision of the First Circuit in *Milward v. Acuity Specialty Products Group, Inc.*, --- F.3d ---, 2011 WL 982385 (1st Cir., Mar. 22, 2011), which is attached as the Supplemental Appendix ("Supp.App.").

Both *Milward* and this case involve diseases (Acute Promyelocytic Leukemia, or APL, in *Milward*, and Parkinson's Disease, or PD, in this case) allegedly triggered by toxic exposures (benzene in *Milward*, manganese in this case). Both illnesses fit within general disease categories long linked to these specific exposures. (Supp.App. 15a; App. 55a). Both opinions make the point that there are scientific theories, but as yet no scientific consensus, concerning individuals' genetic susceptibility to APL and PD and exactly how each disease process is triggered. (Supp.App. 10a; App. 9a-10a). In both lawsuits, experts with undisputed qualifications in relevant fields (Dr. Smith, the plaintiff's toxicologist in *Milward*, and Dr. Carlini, a board-certified neurologist who is Mr. Tamraz's

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[1] 509 U.S. 579 (1983).

[2] 526 U.S. 137 (1999).

[3] 522 U.S. 136 (1997).

treating physician) offered etiology opinions based on scientific theories that indisputably would assist a trier of fact charged with determining whether these toxic exposures caused these diseases. In each appeal, the outcome ostensibly turned on whether the expert's etiology opinion was "reliable" under Rule 702.

Despite striking similarities between the facts and issues in *Milward* and *Tamraz*, their outcomes are utterly irreconcilable. In *Milward*, the district court characterized the expert's etiology opinion as a "hypothesis," albeit a "plausible" one, but ultimately concluded that a "plausible hypothesis" is unreliable under Rule 702. (Supp.App. 29a-30a). The First Circuit reversed, holding that "the [district] court both placed undue weight on the lack of general acceptance of Dr. Smith's conclusions and crossed the boundary between gatekeeper and trier of fact." (*Id.* at 21a).

The majority in *Tamraz* likewise called Dr. Carlini's etiology opinion "a plausible hypothesis," adding, "It may even be right. But it is no more than a hypothesis . . . ." (App. 9a). Unlike the First Circuit, this was enough for the *Tamraz* majority to label Dr. Carlini's opinion unreliable under Rule 702 and to reverse the jury verdict on this basis. (App. 8a ("But the etiological component of [Dr. Carlini's] opinion—the 'manganese-induced' part—was at most a working hypothesis, not admissible scientific 'knowledge.'")). This reversal leaves no doubt that, in the Sixth Circuit, district courts are expected to reject etiology opinions based on scientific hypotheses, as

opposed to scientific consensus. *Milward* sends the opposite signal to district courts in the First Circuit by faulting the district court for taking

> sides on questions that are currently the focus of extensive scientific research and debate—and on which reasonable scientists can clearly disagree. In this, the court overstepped the authorized bounds of its role as gatekeeper.

(Supp.App. 22a).

The First Circuit also faulted the district court for attaching *too much* significance to the fact the expert could not rule out potential explanations for the plaintiff's disease other than the alleged toxic exposure. In *Tamraz*, the majority faulted the district court for attaching *too little* significance to the same fact. (*Compare* Supp.App. 22a ("But the fact that another explanation might be right is not a sufficient basis for excluding Dr. Smith's testimony.") *to* App. 19a (Dr. Carlini's inability "to 'rule out' other possible causes" rendered his testimony speculative and thus inadmissible under Rule 702)). There simply is no common ground between these two approaches to reviewing a district court's "reliability" assessment under Rule 702 and *Daubert*.

According to the majority in *Tamraz*, Dr. Carlini is not permitted to offer a scientific explanation of Mr. Tamraz's illness because he could not "rule out" idiopathic PD, a catch-all term reserved for PD with no known cause. In other words, before Dr. Carlini would be allowed to tell a jury that, to a reasonable degree of scientific certainty, manganese exposure caused Mr. Tamraz's illness, he first would have to be able to

eliminate the possibility that something unknown caused it. Despite the majority's protestations (App. 13a), imposing this logically impracticable burden on an expert amounts to demanding that he state his etiology opinion with absolute certainty, a requirement that is contrary to *Daubert*, 509 U.S. at 590. According to the First Circuit, this type of alleged flaw in an expert's reasoning would "go to the weight of [the expert's] opinion, not its admissibility." (Supp.App. 22a). In *Tamraz*, only the dissent embraced this position. (App. 38a-39a (noting "the majority's newly-minted requirement that scientific testimony must be without flaws" and observing that "[a]t least one other Circuit court" has concluded that "'such arguments go to the weight of the evidence, not its admissibility'" (citing *Campbell v. Metro. Prop. & Cas. Ins. Co.*, 239 F.3d 179, 186 (2d Cir. 2001))).

A side-by-side comparison of the majority opinion in this case and the First Circuit's recent decision in *Milward* leaves no doubt that the outcome of each court's case would have been completely different if decided by the other court. There could be no better illustration of an entrenched Circuit split than this example of two courts of appeals applying *Daubert's* "gatekeeper" function and abuse-of-discretion standard in such strikingly similar situations and reaching such divergent outcomes. Petitioners submit that this case represents the appropriate vehicle for restoring order to the Circuit courts' interpretation of "reliability" under Rule 702 and of *Daubert* and its progeny. Therefore, petitioners respectfully ask the Court to grant the petition and accept jurisdiction.

Respectfully submitted,

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APPENDIX

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SUPPLEMENTAL APPENDIX

United States Court of Appeals
For the First Circuit

No. 09-2270

[Filed March 22, 2011]

BRIAN K. MILWARD AND LINDA J. MILWARD,	)
Plaintiffs, Appellants,	)
v.	)
ACUITY SPECIALTY PRODUCTS GROUP, INC., ET AL.,	)
Defendants, Appellees.	)

APPEAL FROM THE UNITED STATES DISTRICT COURT FOR THE DISTRICT OF MASSACHUSETTS

[Hon. George A. O'Toole, Jr, U.S. District Judge]

Before

Lynch, Chief Judge,
Lipez and Howard, Circuit Judges.

Steve Baughman Jensen, with whom Allen Stewart, P.C., James Gotz, and Kreindler & Kreindler were on brief, for appellants.

Raphael Metzger, Gregory Coolidge, and Metzger Law Group on brief for the Council for Education and Research on Toxins, et al., amici curiae.

Joseph J. Leghorn, with whom Nixon Peabody LLP was on brief, for appellees.

March 22, 2011

LYNCH, Chief Judge. Brian and Linda Milward brought negligence claims against defendant chemical companies alleging that the rare type of leukemia that Brian Milward suffers, Acute Promyelocytic Leukemia (APL), was caused by his routine workplace exposure to benzene-containing products that had been manufactured or supplied by defendants. Milward worked as a refrigeration technician and asserted that he was exposed to benzene from 1973 until the time he filed this complaint and jury demand in October 2007. He had been diagnosed with APL in October 2004.

At defendants' request, the district court bifurcated the suit into two phases. The first phase concerned whether the expert opinion offered by plaintiffs on "general causation" was admissible under Federal Rule of Evidence 702. " 'General causation' exists when a substance is capable of causing a disease." *Restatement (Third) of Torts: Liability for Physical and Emotional*

Harm § 28 cmt. c(3) (2010) ("*Restatement*"). If plaintiffs' expert evidence had been ruled admissible, the second phase would have considered all other issues, including negligence, exposure, and the "specific causation" of Milward's leukemia. " 'Specific causation' exists when exposure to an agent caused a particular plaintiff's disease." *Id.* § 28 cmt. c(4).

This case never reached the second phase. The district court ruled that the testimony of plaintiffs' expert on general causation, Dr. Martyn Smith, was inadmissible under Federal Rule of Evidence 702. The court so ruled after reviewing written statements and materials and conducting a four-day evidentiary hearing in which it heard testimony from plaintiffs' experts Dr. Smith, a toxicologist, and Dr. Carl Cranor, an expert on scientific methodology; and from defendants' experts Dr. David Garabrant, an epidemiologist, Dr. David Pyatt, a toxicologist, and Dr. John Bennett, a pathologist. The district court, in a detailed opinion, ruled that "Dr. Smith's proffered testimony that exposure to benzene can cause APL lacks sufficient demonstrated scientific reliability to warrant its admission under Rule 702." *Milward v. Acuity Specialty Prods. Grp., Inc.*, 664 F. Supp. 2d 137, 140 (D. Mass. 2009). The court entered final judgment for defendants and plaintiffs timely appealed.

The appellate standard of review for Rule 702 rulings is abuse of discretion. *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997). "This standard is not monolithic: within it, embedded findings of fact are reviewed for clear error, questions of law are reviewed de novo, and judgment calls are subjected to classic abuse-of-discretion review." *Ungar v. Palestine Liberation Org.*, 599 F.3d 79, 83 (1st Cir. 2010); *see*

also Baker v. Dalkon Shield Claimants Trust, 156 F.3d 248, 251-52 (1st Cir. 1998) (noting these three dimensions of the abuse of discretion standard in reviewing exclusion of expert testimony).

We reverse the district court's exclusion of Dr. Smith's general causation testimony. *Cf. Ruiz-Troche v. Pepsi Cola of P.R. Bottling Co.*, 161 F.3d 77 (1st Cir. 1998) (reversing exclusion of expert testimony); *Dalkon Shield*, 156 F.3d 248 (same). Dr. Smith's testimony is admissible. We stress that it is up to the jury to decide whether to accept his opinion that exposure to benzene can cause APL--a proposition that plaintiffs must prove by a preponderance of the evidence.

I.

The Supreme Court in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), vested in trial judges a gatekeeper function, requiring that they assess proffered expert scientific testimony for reliability before admitting it.[1] The Court held that Rule 702 displaced the "general acceptance" test of *Frye v. United States*, 293 F. 1013 (D.C. Cir. 1923), under which "the admissibility of an expert opinion or technique turned on its 'general acceptance' *vel non* within the scientific community." *Ruiz-Troche*, 161 F.3d at 80. Under Rule 702:

> If scientific, technical, or other specialized knowledge will assist the trier of fact to

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[1] *Kumho Tire Co. v. Carmichael*, 526 U.S. 137 (1999), clarified that courts have this function with respect to all expert testimony, not just scientific.

> understand the evidence or to determine a fact in issue, a witness qualified as an expert by knowledge, skill, experience, training, or education, may testify thereto in the form of an opinion or otherwise, if (1) the testimony is based upon sufficient facts or data, (2) the testimony is the product of reliable principles and methods, and (3) the witness has applied the principles and methods reliably to the facts of the case.

Fed. R. Evid. 702.

The *Daubert* Court identified four factors that might assist a trial court in determining the admissibility of an expert's testimony: "(1) whether the theory or technique can be and has been tested; (2) whether the technique has been subject to peer review and publication; (3) the technique's known or potential rate of error; and (4) the level of the theory or technique's acceptance within the relevant discipline." *United States v. Mooney*, 315 F.3d 54, 62 (1st Cir. 2002) (citing *Daubert*, 509 U.S. at 593-94). These factors "do not constitute a 'definitive checklist or test.' " *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 150 (1999) (emphasis omitted) (quoting Daubert, 509 U.S. at 593). Given that "there are many different kinds of experts, and many different kinds of expertise," these factors "may or may not be pertinent in assessing reliability, depending on the nature of the issue, the expert's particular expertise, and the subject of his testimony." *Id.*

Exactly what is involved in "reliability" was not and could not have been filled out by *Daubert*. Rather, the answers must come from developing case law in

adjudicating individual controversies. "[T]he question of admissibility 'must be tied to the facts of a particular case.' " *Beaudette v. Louisville Ladder, Inc.*, 462 F.3d 22, 25-26 (1st Cir. 2006) (quoting *Kumho Tire*, 526 U.S. at 150).

Although *Daubert* stated that trial courts should focus "on principles and methodology, not on the conclusions that they generate," *Daubert*, 509 U.S. at 595, the Court subsequently clarified that this focus "need not completely pretermit judicial consideration of an expert's conclusions," *Ruiz-Troche*, 161 F.3d at 81 (citing *Joiner*, 522 U.S. at 146). In *Joiner*, the Court explained that "conclusions and methodology are not entirely distinct from one another" and "nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert." *Joiner*, 522 U.S. at 146. Expert testimony may be excluded if there is "too great an analytical gap between the data and the opinion proffered." *Id.* "[T]rial judges may evaluate the data offered to support an expert's bottom-line opinions to determine if that data provides adequate support to mark the expert's testimony as reliable." *Ruiz-Troche*, 161 F.3d at 81.

This does not mean that trial courts are empowered "to determine which of several competing scientific theories has the best provenance." *Id.* at 85. " *Daubert* does not require that a party who proffers expert testimony carry the burden of proving to the judge that the expert's assessment of the situation is correct." *Id.* The proponent of the evidence must show only that "the expert's conclusion has been arrived at in a scientifically sound and methodologically reliable

fashion." *Id.*; *see also United States v. Vargas*, 471 F.3d 255, 265 (1st Cir. 2006). The object of *Daubert* is "to make certain that an expert, whether basing testimony on professional studies or personal experience, employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Kumho Tire*, 526 U.S. at 152.

So long as an expert's scientific testimony rests upon " 'good grounds,' based on what is known," *Daubert*, 509 U.S. at 590, it should be tested by the adversarial process, rather than excluded for fear that jurors will not be able to handle the scientific complexities, *id.* at 596. "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence." *Id.*; *see also Currier v. United Techs. Corp.*, 393 F.3d 246, 252 (1st Cir. 2004).

II.

It is uncontested that Dr. Smith's opinion about the causal link between benzene and APL satisfies certain requirements of Rule 702. His opinion would "assist the trier of fact to understand the evidence or to determine a fact in issue." Fed. R. Evid. 702. And Dr. Smith is "a witness qualified as an expert by knowledge, skill, experience, training, or education." *Id.* He is acknowledged as a leading expert on the study of the toxic effects of chemicals and drugs on the human body, with particular emphasis on the mechanisms by which benzene and its metabolites cause damage to both cells and the human organism as a whole. The research in Dr. Smith's laboratory, which is funded by the National Institutes of Health, focuses

on the causes of leukemia and lymphoma, and he has authored or co-authored over 215 articles in peer-reviewed journals in the field of toxicology.

The question before us is whether the district court abused its discretion in concluding that the other requirements of Rule 702, concerning the reliability of Dr. Smith's opinion, were not met. We will first discuss some basic facts about leukemia, the weight of the evidence methodology, and Dr. Smith's use of that methodology, and we will then turn to an evaluation of the district court's ruling.

Leukemia is a cancer of the blood cells. There are different types of leukemia, which are generally classified in two ways. The first classification is between leukemia's acute and chronic forms: acute leukemia is characterized by a rapid increase in the number of immature blood cells, while chronic leukemia is characterized by the excessive buildup of relatively mature but abnormal white blood cells. The second classification is between the types of stem cells affected: leukemia can be either "myeloid" or "lymphoid." Combining these two classifications provides a total of four main categories of leukemia: acute myeloid leukemia (AML); chronic myeloid leukemia (CML); acute lymphoid leukemia (ALL); and chronic lymphoid leukemia (CLL).[2] Within each of these categories, there are typically several subcategories.

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[2] There are also some types of leukemia that are considered to be outside of this four-part classification scheme.

The general category of AML can be subdivided in more than one way. Under the common French-American-British classification system used by the parties, subtypes are classified morphologically according to the degree of differentiation along different cell lines and the extent of cell maturation. This classification system identifies subtypes by convention as M0 through M7.[3]

Brian Milward's leukemia, APL, is subtype M3 and is an extremely rare disease. APL accounts for only five to ten percent of all cases of AML, which is itself rare, with an annual incidence of 3.5 cases per 100,000 people. APL is characterized by a deficiency of mature blood cells in the myeloid cell line and an excess of immature cells called promyelocytes.

APL is in part caused by the chromosomal translocation of a gene known as the retinoic acid receptor-alpha gene (RARα) on chromosome 17.[4]

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[3] The World Health Organization has adopted a different classification system that utilizes not only morphological characteristics, but also genetic, immunophenotypic, biologic, and clinical characteristics to define specific disease entities that have clinical and biological relevance. *See generally* James W. Vardiman et al., *The World Health Organization (WHO) Classification of the Myeloid Neoplasms*, 100 Blood 2292 (2002).

[4] In approximately 95% of cases of APL, RARα is involved in a reciprocal translocation with the promyelocytic leukemia gene (PML) on chromosome 15--a translocation denoted as t(15;17)(q22;q12)--which creates a fusion gene known as PML-RARα. In

Although APL and the other subtypes of AML have been the subject of extensive research, there is not yet a scientific consensus as to the causes of the genetic translocation that induces APL.

Dr. Smith's opinion is that what is known about both AML and APL supports the inference that exposure to benzene can cause APL. He reached this opinion using a "weight of the evidence" methodology in which he considered five lines of evidence drawn from the peer-reviewed scientific literature on leukemia and benzene. We first discuss the reliability of this methodology in general, and then turn to Dr. Smith's application of it.

A. The Reliability of the Weight of the Evidence Methodology

Dr. Smith's opinion was based on a "weight of the evidence" methodology in which he followed the guidelines articulated by world-renowned epidemiologist Sir Arthur Bradford Hill in his seminal methodological article on inferences of causality. *See* Arthur Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 Proc. Royal Soc'y Med. 295 (1965).

Hill's article explains that one should not conclude that an observed association between a disease and a feature of the environment (e.g., a chemical) is causal without first considering a variety of "viewpoints" on the issue. These viewpoints include: the strength or

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the remaining cases of APL, RARα translocates and fuses with one of four other genes.

frequency of the association; the consistency of the association in varied circumstances; the specificity of the association; the temporal relationship between the disease and the posited cause; the dose response curve between them; the biological plausibility of the causal explanation given existing scientific knowledge; the coherence of the explanation with generally known facts about the disease; the experimental data that relates to it; and the existence of analogous causal relationships. *See id.* at 295-99.[5]

Although Hill identified nine viewpoints, it is generally agreed that this list is not exhaustive and that no one type of evidence must be present before causality may be inferred. For example, when a group from the National Cancer Institute was asked to rank the different types of evidence, it concluded that "[t]here should be no such hierarchy."[6] Michele Carbon

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[5] *See also* Sheldon Krimsky, *The Weight of Scientific Evidence in Policy and Law*, 95 Am. J. Pub. Health S129, S129 (2005) (explaining that the term "weight of the evidence" is used "to characterize a process or method in which all scientific evidence that is relevant to the status of a causal hypothesis is taken into account").

[6] This point was also emphasized by Hill, who cautioned in his article:

> None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a sine qua non. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question--is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect?

Austin Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 Proc. Royal Soc'y Med. 295, 299 (1965).

et al., *Modern Criteria to Establish Human Cancer Etiology*, 64 Cancer Res. 5518, 5522 (2004); *see also* Sheldon Krimsky, *The Weight of Scientific Evidence in Policy and Law*, 95 Am. J. Pub. Health S129, S130 (2005).

This "weight of the evidence" approach to making causal determinations involves a mode of logical reasoning often described as "inference to the best explanation," in which the conclusion is not guaranteed by the premises.[7] *See Bitler v. A.O. Smith Corp.*, 391 F.3d 1114, 1124 n. 5 (10th Cir. 2004). As explained by plaintiffs' expert on methodology Dr. Cranor, Distinguished Professor of Philosophy at the University of California, Riverside, inference to the best explanation can be thought of as involving six general steps, some of which may be implicit. The scientist must (1) identify an association between an exposure and a disease, (2) consider a range of plausible explanations for the association, (3) rank the rival explanations according to their plausibility, (4) seek additional evidence to separate the more plausible from the less plausible explanations, (5) consider all of the relevant available evidence, and (6) integrate the evidence using professional judgment to come to a conclusion about the best explanation.

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[7] "Unlike a logical inference made by deduction where one proposition can be logically inferred from other known propositions, and unlike induction where a generalized conclusion can be inferred from a range of known particulars, inference to the best explanation--or 'abductive inferences'--are drawn about a particular proposition or event by a process of eliminating all other possible conclusions to arrive at the most likely one, the one that best explains the available data." *Bitler v. A.O. Smith Corp.*, 391 F.3d 1114, 1124 n. 5 (10th Cir. 2004).

In this mode of reasoning, the use of scientific judgment is necessary. "No algorithm exists for applying the Hill guidelines to determine whether an association truly reflects a causal relationship or is spurious." *Restatement* § 28 cmt. c(3). Because "[n]o scientific methodology exists for this process . . . reasonable scientists may come to different judgments about whether such an inference is appropriate." *Id.* § 28 reporters' note cmt. c(4).

The fact that the role of judgment in the weight of the evidence approach is more readily apparent than it is in other methodologies does not mean that the approach is any less scientific. No matter what methodology is used, "an evaluation of data and scientific evidence to determine whether an inference of causation is appropriate requires judgment and interpretation." *Id.* § 28 cmt. c(1).[8] The use of judgment in the weight of the evidence methodology is similar to that in differential diagnosis, *see Cruz v. Bridgestone/Firestone N. Am. Tire, LLC*, 388 Fed. Appx. 803, 806-07 (10th Cir. 2010) (explaining that differential analysis in general is best characterized as a process of reasoning to the best explanation), which we have repeatedly found to be a reliable method of medical diagnosis, *see Granfield v. CSX Transp., Inc.*,

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[8] The fact that epidemiology relies on statistical methods does not avoid the use of judgment, as "[e]ven sampling error, which is analyzed using quantitative statistical methods, only provides a range of outcomes (associations) that might have been produced by sampling error even if there is no association between the agent and disease. Thus, interpreting the results of epidemiologic studies requires informed judgment and is subject to uncertainty." *Restatement (Third) of Torts: Liability for Physical and Emotional Harm* § 28 reporters' note cmt. c(3) (2010).

597 F.3d 474, 486 (1st Cir. 2010); *Dalkon Shield*, 156 F.3d at 253.

Defendants argue that "regardless of its level of acceptance in the scientific community, a pure 'weight of the evidence' approach like that utilized by Dr. Smith . . . is hardly the type of reliable scientific evidence contemplated by *Daubert*."[9] No serious argument can be made that the weight of the evidence approach is inherently unreliable. Rather, admissibility must turn on the particular facts of the case. *See, e.g.*, *Cruz*, 388 Fed. Appx. at 807 (explaining that expert testimony based on "inference to the best explanation" may be admissible, but that there was no

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[9] Defendants draw our attention to a Fifth Circuit case, excluding testimony based on a weight of the evidence methodology, in which the court explained:

> We are also unpersuaded that the 'weight of the evidence' methodology these experts use is scientifically acceptable for demonstrating a medical link between Allen's EtO exposure and brain cancer. Regulatory and advisory bodies such as IARC, OSHA and EPA utilize a 'weight of the evidence' method to assess the carcinogenicity of various substances in human beings and suggest or make prophylactic rules governing human exposure. This methodology results from the preventive perspective that the agencies adopt in order to reduce public exposure to harmful substances. The agencies' threshold of proof is reasonably lower than that appropriate in tort law. . . .

Allen v. Pa. Eng'g Corp., 102 F.3d 194, 198 (5th Cir. 1996). However, the Fifth Circuit did not, as defendants contend, hold "that the 'weight-of-the evidence' approach is per se unreliable." Rather, the court rejected its use in that case--a case in which it found that the experts' conclusion was "at best weakly supported, if not contradicted, by the evidence on which they rely," and in which the experts "all declined to say that they would subject their findings to the test of peer review for publication." *Id.*

error in the district court's finding that the expert's specific theory did not have sufficient scientific support). Here, the question is whether Dr. Smith, in reaching his opinion, applied the methodology with "the same level of intellectual rigor" that he uses in his scientific practice. *Kumho Tire*, 526 U.S. at 152.

B. Dr. Smith's Application of the Methodology

In concluding that the weight of the evidence supported the conclusion that benzene can cause APL, Dr. Smith relied on his knowledge and experience in the field of toxicology and molecular epidemiology and considered five bodies of evidence drawn from the peer-reviewed scientific literature on benzene and leukemia.

First, Dr. Smith considered the near-consensus among governmental agencies, experts, and active researchers in the field that benzene can cause AML as a class. The existence of this causal connection has been established since the late 1970s. *See* Bernard D. Goldstein & Gisela Witz, *Benzene*, *in Environmental Toxicants: Human Exposures and Their Health Effects* 459, 478 (Morton Lippmann ed., 3rd ed. 2009). Dr. Smith noted that epidemiological studies have found a statistically significant increased incidence of AML in benzene-exposed workers and have identified a dose-response relationship.

Second, Dr. Smith considered evidence concerning the etiology, or origins, of leukemia indicating that all types of AML derive from a genetically damaged pluripotent stem cell. Dr. Smith referred to a recent peer-reviewed article that provided a review of the current literature and reported numerous studies

demonstrating that both AML and CML are stem cell diseases. He cited peer-reviewed studies finding that in the APL and Core Binding Factor (CBF) subtypes of AML, as well as in CML, the stem cell mutation is often in part caused by a chromosomal translocation. He also cited evidence that APL and CBF share common genetic susceptibility factors, common risk factors, and the same incidence pattern occurring at a constant incidence with age after age 20. Dr. Smith concluded that the best explanation for this evidence is that all AMLs, including APL, have a common etiology.[10]

Third, Dr. Smith considered toxicology studies establishing that metabolites of benzene cause significant chromosomal damage at the stem cell level in the bone marrow--the type of damage that is known to cause APL and other types of AML.[11] He also cited peer-reviewed work published by his lab showing that leukemia cases associated with benzene exposure are more likely to contain clonal chromosome aberrations than leukemias arising in the general population.

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[10] Defendants' experts questioned Dr. Smith's conclusion that all of the subtypes of AML have a common etiology. However, on cross examination in the district court *Daubert* hearing, defendants' expert Dr. Pyatt agreed with the statement that "there are a group of reasonable scientists who reasonably believe that all forms of AML arise from the same progenitor cell" and stated that Dr. Smith's opinion was "consistent with most of the evidence." Defendants' expert Dr. Bennett likewise agreed that "reasonable scientists can and do" agree with Dr. Smith.

[11] Defendants' expert Dr. Bennett agreed that "there have been innumerable studies that demonstrate that benzene actually works at multiple levels to create damage to the DNA structure of this hematopoietic stem cell."

Fourth, Dr. Smith considered two sets of studies concerning the inhibition of a cellular enzyme known as topoisomerase II (or "topo II") that is essential for the maintenance of proper chromosome structure and segregation. One set of studies--including both test tube and animal studies--has established that two benzene metabolites are catalytic inhibitors of topo II. A second set of studies has established that a variety of chemotherapeutic agents that are catalytic inhibitors of topo II cause APL.[12] Dr. Smith explained that taken together, these studies provided evidence of a known biological mechanism by which exposure to benzene could cause APL.

Fifth, Dr. Smith considered the small set of epidemiological studies that provide data on the relationship between benzene exposure and subtypes of AML.[13] He concluded that the evidence showed an

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[12] Dr. Smith cited a long list of peer-reviewed publications and quoted a recent authoritative paper in a prominent journal stating that "[t]herapy-related acute promyelocytic leukemia (t-APL) with the t(15;17) translocation is a well recognized complication of cancer treatment with agents targeting topoisomerase II." Syed Khizer Hasan et al., *Molecular Analysis of t(15;17) Genomic Breakpoints in Secondary Acute Promyelocytic Leukemia Arising After Treatment of Multiple Sclerosis*, 112 Blood 3383, 3383 (2008). Defendants' hematopathologist, Dr. Bennett, acknowledged that chemotherapeutic compounds that inhibit topo II can cause APL.

[13] He considered a multi-center Chinese case-control study of 1257 cases of leukemia in which there was a 40% increased risk of APL in benzene-exposed workers; a cohort study of 74,828 workers exposed to benzene in China in which APL was the most common form of AML diagnosed; a multi-center Italian case-control study of 38 cases of APL that showed a strong association between APL and shoe-making, an industry that had for many years used

increased risk factor for APL, consistent with causality, and provided no grounds for concluding otherwise.

Dr. Smith explained that taking into account all of the evidence described above--the fact that benzene causes AML as a class, that all subtypes of AML likely have a common etiology, that benzene is known to cause the type of chromosomal damage characteristic of APL, that benzene is known to inhibit an enzyme whose inhibition is known to cause APL, and that APL has been reported in benzene-exposed workers in a number of epidemiological studies--he reached the opinion that the weight of the evidence supports the conclusion that benzene exposure is capable of causing APL. Dr. Smith's opinion rests on a scientifically sound and methodologically reliable foundation, as is required by *Daubert*.

III.

In finding Dr. Smith's opinion inadmissible under Rule 702, the district court relied on (a) its evaluation of the mechanistic and epidemiological evidence on which Dr. Smith based his opinion, and (b) its understanding of the scientific concept of "biological plausibility" as used by Dr. Smith when he explained his conclusions. As we explain below, on both of these points, the district court erred. In the end, the court's exclusion of the testimony was based on its evaluation of the weight of the evidence, which is an issue that is the province of the jury, and on its misperception of

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benzene as an adhesive; and several case reports of APL in benzene-exposed workers.

the methodology and analysis that provided the basis for Dr. Smith's opinion.

A. The Evidentiary Basis of Dr. Smith's Opinion

1. Mechanistic Evidence

The district court's exclusion of Dr. Smith's testimony was based to a significant extent on its rejection of what it took to be his three key subsidiary conclusions regarding the weight of the mechanistic evidence. We briefly summarize the court's analysis on these points before turning to our discussion of the ways in which the court erred in its analysis.

First, the court held that there was insufficient evidence to support Dr. Smith's opinion that all subtypes of AML likely have a common etiology. The court reasoned that the "clear differences" among AML subtypes--in particular, APL's unique response to certain types of therapy, and the subtypes' different chromosomal abnormalities--made "a broad extrapolation from AML generally to APL specifically" inappropriate.[14] *Milward*, 664 F. Supp. 2d at 144. The court also noted that a series of recent studies had "led investigators to think that the 'leukemic stem cell' may exist in more mature, differentiated cell lines," such

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[14] Dr. Smith's supplemental report makes it clear that this is something on which reasonable scientific disagreement is possible. He explained that in his view, defendants' experts erred in concluding that the fact that APL is therapeutically unique means that it is also etiologically unique. Identifying the biological mechanism that made APL therapeutically unique--the sensitivity of the PML-RARα fusion gene to retinoic acid and arsenic--he explained that this was "irrelevant" to APL's etiology.

that "the 'leukemic stem cell' may not be a stem cell in the usual sense, but rather a differentiated cell that has somehow acquired the ability to reproduce itself, as a stem cell can." *Id.* at 145. If the various AML subtypes did not arise from the same progenitor or stem cell, the court reasoned, they might well not share a common etiology.[15] Finally, the court emphasized that there was "no scientific consensus" on this issue, and that the question of when the key chromosomal translocation occurs was considered by researchers to be "*a question that remains unanswered in the APL field*." *Id.* (quoting S. Wojiski et al., *PML-RARα Initiates Leukemia by Conferring Properties of Self-Renewal to Committed Promyelocytic Progenitors*, 23 Leukemia 1462, 1469 (2009) (emphasis added)) (internal quotation marks omitted).

Second, the court held that what was known about the types of chromosomal translocations caused by benzene did not offer sufficient support for Dr. Smith's opinion that it is biologically plausible that benzene causes the characteristic t(15;17)(q22;q12) translocation seen with APL. The court explained that this opinion would be warranted if benzene's impact on chromosomes were randomly experienced, but it noted that a paper co-authored by Dr. Smith concluded that "benzene can initiate or promote leukemia induction by a *nonrandom* selective effect" on specific

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[15] In the *Daubert* hearing, Dr. Smith made it clear that he had considered the key paper cited by the district court on this point. He noted that it was based on studies in "mice using a highly artificial system," and he explained that even if the mutation could occur at a later point in differentiation as indicated by this paper, "it doesn't mean that it has to occur only in that compartment."

chromosomes. *Id.* at 147 (emphasis added). This defeated "the generalization that because . . . benzene causes damage to some chromosomes, it is 'biologically plausible' that it causes damage to other chromosomes." *Id.*

Third, the court held that there was insufficient evidence to support the inference that benzene metabolites inhibit topo II in such a way as to cause the chromosomal translocation seen in cases of APL. The court's conclusion was in part based on evidence that "[t]here are different classes of topo II inhibitors and the different classes have been associated with different AML subtypes." *Id.* Highlighting one article's finding that leukemias induced by benzene do not appear to exhibit the defining characteristics associated with four other classes of topo II inhibitors, id. at 148, the court held that to "the extent that Dr. Smith's opinion rests on the proposition that all topo II inhibitors act similarly to cause a similar effect, then, it does not appear to be based on reliable scientific knowledge," *id.* at 147.

In reaching these three conclusions about some of the evidence on which Dr. Smith based his opinion, the court both placed undue weight on the lack of general acceptance of Dr. Smith's conclusions and crossed the boundary between gatekeeper and trier of fact.

Although general acceptance is still a relevant consideration under *Daubert*, the court's demands went too far. *Cf. Smith v. Ford Motor Co.*, 215 F.3d 713, 721 (7th Cir. 2000) (reversing district court that had treated lack of peer review as dispositive grounds for excluding expert opinion). On the question of the origins of APL, for example, the court explained that

in the absence of consensus about the target cell for the leukemic mutation, Dr. Smith's opinion that all forms of AML likely share a common origin was "at best a plausible hypothesis." *Milward*, 664 F. Supp. 2d at 146. The court explained that the fact that "other plausible hypotheses . . . might be true as well, including the hypothesis that the genetic mutation that leads to APL occurs in relatively mature cells," meant that Dr. Smith's opinion was not "based on sufficient facts and data to be accepted as a reliable scientific conclusion." *Id.*; *see also id.* at 148 (focusing on lack of consensus as to the topo II question). But the fact that another explanation might be right is not a sufficient basis for excluding Dr. Smith's testimony. "Lack of certainty is not, for a qualified expert, the same thing as guesswork." *Primiano v. Cook*, No. 06-15563, 2010 WL 1660303, *5 (9th Cir. Apr. 27, 2010).

In addition, the alleged flaws identified by the court go to the weight of Dr. Smith's opinion, not its admissibility. There is an important difference between what is *unreliable* support and what a trier of fact may conclude is *insufficient* support for an expert's conclusion.

The court's analysis repeatedly challenged the factual underpinnings of Dr. Smith's opinion, and took sides on questions that are currently the focus of extensive scientific research and debate--and on which reasonable scientists can clearly disagree. In this, the court overstepped the authorized bounds of its role as gatekeeper. "The soundness of the factual underpinnings of the expert's analysis and the correctness of the expert's conclusions based on that analysis are factual matters to be determined by the

trier of fact." *Smith*, 215 F.3d at 718. "When the factual underpinning of an expert's opinion is weak, it is a matter affecting the weight and credibility of the testimony--a question to be resolved by the jury." *Vargas*, 471 F.3d at 264 (quoting *Int'l Adhesive Coating Co. v. Bolton Emerson Int'l*, 851 F.2d 540, 545 (1st Cir. 1988)) (internal quotation marks omitted); *see also Quiet Tech. DC-8, Inc. v. Hurel-Dubois UK Ltd.*, 326 F.3d 1333, 1345 (11th Cir. 2003); *Amorgianos v. Nat'l R.R. Passenger Corp.*, 303 F.3d 256, 267 (2d Cir. 2002).

Of course, following *Joiner*, a "district court properly may exclude expert testimony if the court concludes too great an analytical gap exists between the existing data and the expert's conclusion." *Kennedy v. Collagen Corp.*, 161 F.3d 1226, 1230 (9th Cir. 1998). Here, however, "the gap was of the district court's making." *Id.* Dr. Smith's opinion was based on a reliable methodology and substantial evidence that he carefully explained. The questions that the court posed were sensible ones, but ones for the jury to resolve.

At times, the court's error in excluding Dr. Smith's testimony derived from a mistake in its understanding of the weight of the evidence methodology employed by Dr. Smith. The court treated the separate evidentiary components of Dr. Smith's analysis atomistically, as though his ultimate opinion was *independently* supported by each. For example, the court referred to "Dr. Smith's opinion that *because* benzene metabolites inhibit topo II and because some classes of topo II inhibitors appear to have a causal relationship to APL, *therefore* benzene has a causal relationship to APL." *Milward*, 664 F. Supp. 2d at 148 (emphasis added). This overstates Dr. Smith's conclusion as to the topo II

evidence, and is indicative of an error in the court's understanding of the nature of Dr. Smith's analysis.

In Dr. Smith's weight of the evidence approach, no body of evidence was itself treated as justifying an inference of causation. Rather, each body of evidence was treated as grounds for the subsidiary conclusion that it would, if combined with other evidence, support a causal inference. The district court erred in reasoning that because no one line of evidence supported a reliable inference of causation, an inference of causation based on the totality of the evidence was unreliable. *Cf. NutraSweet Co. v. X-L Eng'g Co.*, 227 F.3d 776, 789 (7th Cir. 2000) (holding that an expert's reliance on individual pieces of evidence, insufficient in themselves to prove a point, "did not render his opinion speculative").[16] The hallmark of the weight of the evidence approach is reasoning to the best explanation for all of the available evidence. *Cf. Dalkon Shield*, 156 F.3d at 253 (reversing district court's exclusion of expert testimony as "guesswork" or without "basis" when testimony was based on differential diagnosis and there was no showing that any one of the expert's premises was "so faulty that it could not even be tendered to the jury for its consideration"); *see also Hardyman v. Norfolk & W. Ry. Co.*, 243 F.3d 255, 261 (6th Cir. 2001).

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[16] As a general evidentiary matter, "individual pieces of evidence, insufficient in themselves to prove a point, may in cumulation prove it," and "a piece of evidence, unreliable in isolation, may become quite probative when corroborated by other evidence." *Bourjaily v. United States*, 483 U.S. 171, 179-80 (1987).

2. Epidemiological Evidence

As to the epidemiological evidence on which Dr. Smith based his opinion in part, the court held that the published articles on which Dr. Smith relied did not support his opinion, and that in any event, the evidence was not statistically significant. On these grounds, the court rejected Dr. Smith's conclusion that the available epidemiological evidence offered some support for an inference of causation.

In concluding that the papers cited by Dr. Smith did not support his opinion, the court reasoned that "Dr. Garabrant convincingly demonstrated, especially with respect to the Golomb and Travis papers, that Dr. Smith's conclusions that there was a positive association between exposure to benzene and APL were based on faulty calculations of odds ratios." *Milward*, 664 F. Supp. 2d at 149. An odds ratio represents the difference in the incidence of a disease between a population that has been exposed to benzene and one that has not. In Dr. Garabrant's opinion, Dr. Smith should have used the incidence rate of APL for the general population as a baseline, rather than the rate for non-benzene-exposed workers. In the *Daubert* hearing and in his supplemental report, however, Dr. Smith explained that he disagreed with Dr. Garabrant on this point, but that in any event, the odds ratio was still elevated, consistent with an inference of causation. Where, as here, both experts' opinions are supported by evidence and sound

scientific reasoning, the question of who is right is a question for the jury.[17]

The court explained, however, that even if "some of the data reported in the various studies could be properly understood to suggest a positive association, the findings are not statistically significant," *id.*, and that although "epidemiological evidence is not always essential," the defendants were "correct that sound epidemiological studies are ordinarily needed to confirm, by consistent observation, an hypothesis of causation," *id.* at 148.

In context, the district court read too much into the paucity of statistically significant epidemiological studies. The absence of peer-reviewed epidemiological studies does not, as defendants contend, make it "almost impossible" for Dr. Smith's opinion to be admissible. Epidemiological studies are not per se required as a condition of admissibility regardless of context. *See Rider v. Sandoz Pharm. Corp.*, 295 F.3d 1194, 1198 (11th Cir. 2002) ("It is well-settled that while epidemiological studies may be powerful evidence of causation, the lack thereof is not fatal to a plaintiff's case."); *Restatement* § 28 reporters' note cmt. c(3) (listing federal circuit cases holding that epidemiological data is not necessary). Nor are such

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[17] The court also rejected Dr. Smith's analysis of the epidemiological evidence on the grounds that "none of the studies purports to give direct support to the proposition that benzene causes APL." *Milward v. Acuity Specialty Prods. Grp., Inc.*, 664 F. Supp. 2d 137, 148 (D. Mass. 2009). Yet Dr. Smith did not claim that the studies provided direct support. Rather, his characterization of his methodology makes clear that he was using them as indirect support.

studies treated as always essential in the relevant scientific communities.

To be clear, this is not a situation in which the available epidemiological studies found that there is no causal link, or even one in which no cases of APL were found among benzene-exposed workers. *Cf. Norris v. Baxter Healthcare Corp.*, 397 F.3d 878, 882 (10th Cir. 2005) (holding that epidemiological studies are not required to prove causation, but that a substantial body of epidemiological evidence challenging causation cannot be ignored); *Allen v. Pa. Eng'g Corp.*, 102 F.3d 194, 197 (5th Cir. 1996) (finding it significant that "numerous reputable epidemiological studies covering in total thousands of workers" indicated that there was no causation).

Rather, this is a case in which the few studies that differentiate between AML and APL do not offer conclusive statistically significant evidence either way, in part because the rarity of APL makes it nearly impossible to perform a large enough study.[18] Dr. Smith estimated that in order to obtain statistically significant results, one would need hundreds of thousands of highly exposed workers, the same number of controls, and millions of dollars in funding. The court erred in treating the lack of statistical

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[18] The difficulty of performing such a study is not contested by defendants, and it has even been expressly affirmed in the scientific literature. *See* Dan Douer, *The Epidemiology of Acute Promyelocytic Leukaemia*, 16 Best Prac. & Res. Clinical Haematology 357, 358 (2003) ("It is difficult to perform epidemiological studies in AML subtypes classified according to cytogenetic abnormalities owing to the small number of patients within each subgroup.").

significance as a crucial flaw. *See Collagen Corp.*, 161 F.3d at 1229 (finding that the district court placed too much emphasis on lack of epidemiological studies where such studies "would be almost impossible to perform"); *see also Primiano*, 2010 WL 1660303, at *5-6 (noting that peer-reviewed studies are not necessary, especially when there are good reasons why such studies have not been performed). Under these circumstances, the court erred in holding that "Dr. Smith's attempt to support his conclusion with data that concededly lacks statistical significance" was "a deviation from sound practice of the scientific method" that provided grounds for exclusion. Milward, 664 F. Supp. 2d at 149.

The court's evaluation of the epidemiological evidence is also in tension with the weight of the evidence methodology. Dr. Smith explained that his citation to epidemiological data was meant to challenge the theory that benzene exposure could not cause APL, and to highlight that the limited data available was consistent with the conclusions that he had reached on the basis of other bodies of evidence. He stated that "[i]f epidemiologic studies of benzene-exposed workers were devoid of workers who developed APL, one could hypothesize that benzene does not cause this particular subtype of AML." The fact that, on the contrary, "APL is seen in studies of workers exposed to benzene where the subtypes of AML have been separately analyzed and has been found at higher levels than expected" suggested to him that the limited epidemiological evidence was at the very least consistent with, and suggestive of, the conclusion that benzene can cause APL.

The court rejected Dr. Smith's reasoning, stating that a " 'suggestion' may give rise to a plausible hypothesis, but not a reliable inference." *Milward*, 664 F. Supp. 2d at 149. But as noted above, this is inconsistent with the scientifically accepted methodology employed by Dr. Smith. Dr. Smith did not infer causality from this suggestion alone, but rather from the accumulation of multiple scientifically acceptable inferences from different bodies of evidence.

B. The Concept of "Biological Plausibility"

The district court also erred in its apprehension of the scientific concept of biological plausibility and its place in Dr. Smith's analysis. The concept of biological plausibility, which numbers among the nine Hill viewpoints, asks whether the hypothesized causal link is credible in light of what is known from science and medicine about the human body and the potentially offending agent. At two places in the court's analysis, it conflated the scientific question of biological plausibility with the legal question of probability.

In the court's discussion of the epidemiological evidence, it stated that even if the evidence " 'suggests' a causal relationship," providing support for Dr. Smith's opinion regarding biological plausibility, a "plausible hypothesis" is not a "reliable inference" and is therefore inadmissible. *Id.* Here, the court not only misconstrued the concept of biological plausibility by equating it with a merely plausible or possible hypothesis, but also misconstrued the concept's role in Dr. Smith's analysis by assuming that Dr. Smith treated the criteria as sufficient grounds for inferring causality (rather than as one consideration that entered into his weighing of the evidence).

The court made a similar error in its conclusion, where it stated:

> While Dr. Smith's hypotheses are, to use his term, "plausible," they remain hypotheses, the validity of which has not been reliably established. . . . [T]he sum of Dr. Smith's testimony, fairly understood, is that benzene *might be* a cause of APL.

Id. Again, the district court misunderstood Dr. Smith to be saying that causation is possible rather than probable. The sum of Dr. Smith's testimony was not merely that it is possible, or even biologically plausible, that benzene causes APL. Rather, the sum of his testimony was that a weighing of the Hill factors, including biological plausibility, supported the inference that the association between benzene exposure and APL is genuine and causal.

IV.

The record clearly demonstrates that Dr. Smith's opinion was based on an analysis in which he employed the "same level of intellectual rigor" that he employs in his academic work. *Kumho Tire*, 526 U.S. at 152. In excluding Dr. Smith's testimony, the district court did not properly apply *Daubert* and exceeded the scope of its discretion. We reverse the district court's judgment for the defendants and its exclusion of Dr.

Smith's testimony, and we remand for proceedings consistent with this opinion.[19]

So ordered.

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[19] We wish to acknowledge the able briefing of the issues by the parties and amici.